THE BOSTON
Medical and Surgical
JOURNAL

VOLUME 195 SEPTEMBER 30, 1926 NUMBER 14

ORIGINAL ARTICLES

RELATIONSHIP BETWEEN GASTRIC ULCER AND CARCINOMA OF STOMACH*

BY JOHN S. LAWRENCE, M.D.,† AND A. V. BOCK, M.D.

THE incidence of carcinoma of the stomach in gastric ulcer has caused considerable discussion during the past decade. In general there have been two principal schools in this controversy. The Mayo Clinic, as represented by MacCarty and others, and Moynihan have been the main supporters of the theory that a high percentage of cases of malignancy of the stomach are preceded by ulcer. Among others, Ewing, of this country, and Dibble, of England, have strongly upheld the view that only a small percentage of gastric ulcers are transformed into carcinoma of the stomach. MacCarty[1] in 1922 said, "Most chronic gastric ulcerations with a diameter greater than 2.5 centimeters are cancerous." Out of one hundred and fifty-three cases of undoubted carcinoma of the stomach, Wilson and MacCarty[2] have found one hundred and nine (71 per cent.) which presented sufficient gross and microscopic evidence of previous ulcer to warrant placing them in a group labelled "carcinoma developing on preceding ulcer." Eleven other cases (7 per cent.) showed considerable evidence of preceding ulcer. Recently MacCarty[3] has made the following statement: "I have never seen an early carcinoma of the stomach that was not associated with an ulcer. These being facts in a large experience I naturally am suspicious of any *chronic* gastric ulcer." In his Hunterian Lecture in 1923 Moynihan[4] made the following statement: "Of the cases of cancer of the stomach that I see upon the operation table a steady average of two out of three give a past history that is extremely suggestive of gastric ulcer." Additional statements in this connection have been made by William J. Mayo[5] and MacCarty and Broders[6].

Ewing[7] holds a view very much opposed to that expressed by the above writers. In 1918 he stated that "the cancerous transformation of peptic ulcer is rather infrequent and probably does not exceed the incidence of five per cent. originally established." Dibble[8] in a study of one hundred and sixty-four stomachs arrived at very similar conclusions. Morley[9] in 1923 stated that a patient with chronic simple ulcer of the stomach was little, if at all, more liable to cancer than a healthy individual. Taylor and Miller[10] in a clinical study of one hundred and eighty-two cases of carcinoma of the stomach lend support to the views of those who believe in a low incidence of carcinoma in ulcers of the stomach.

It is interesting to note what Osler and McCrae had to say on this subject in 1900[11]. After a study of one hundred and fifty cases of cancer of the stomach at the Johns Hopkins Hospital they made the following statement: "In only four of our series was there a history which pointed to gastric ulcer, and in none of these could such a condition be positively diagnosed." After comparing the incidence of stomach troubles in a series of hospital cases with that in the series of cases with carcinoma of the stomach they said: ". . . We may conclude so far as the figures show anything, that the victims of chronic dyspepsia and the various forms of gastritis are not more prone to malignant disease than other individuals."

The present report is based on a study of ninety-eight cases of carcinoma of the stomach and forty-eight cases of ulcer of the stomach. Of the cases of cancer, sixty-eight (69 per cent.) were operated upon. A positive microscopic examination was obtained in approximately one-quarter (24.5 per cent.) of the cases in the cancer series. Forty out of the forty-eight cases (83 per cent.) of ulcer of the stomach came to operation. Approximately two-fifths (37.5 per cent.) of these cases received microscopic examination with a positive report of ulcer. It will be seen therefore that a majority of these cases were confirmed by operation and that an appreciable number was further confirmed by pathological examination.

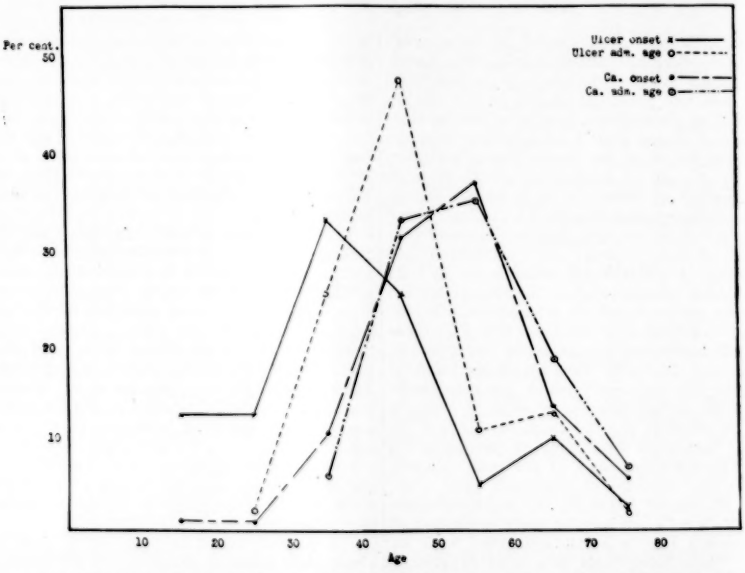
The attempt has been made to compare the clinical findings in the two conditions in these cases. The principal points brought out by this comparison may be considered as follows:

*From the Medical and Surgical Services of the Massachusetts General Hospital.
†Edward Hickling Bradford Fellow in Medical Research, Harvard Medical School.

AGE

Figure 1 shows the age of onset and admission age of the cases in each series. It will be seen that the admission age for ulcer reaches its peak in the fifth decade and that for carcinoma in the sixth decade. With regard to the age of onset it is found that the highest percentage of cases of carcinoma of the stomach are in the sixth decade, whereas with the ulcer cases the peak is reached in the third decade. In other words, the majority of cases of ulcer of the stomach have their initial symptoms about twenty years earlier than the majority of subjects with gastric malignancy.

FIGURE 1



DURATION

The average duration of symptoms prior to entry to the hospital was approximately eight years (7.97 years) for forty cases of gastric ulcer in our series. In eight cases we could not be sure of the duration and so these were not included. The shortest duration which was found for ulcer was one month. There were two such cases. There were seven cases of ulcer with a duration of less than six months.

Ninety-six cases of carcinoma of the stomach had an average duration of approximately two and a half years (2.4 years). This included, however, five cases which had had mild gastro-intestinal complaints such as "weak stomach," "inflammation of the bowels" and "heart-burn" for years. There is, of course, the possibility of ulcer having antedated carcinoma in these cases but it would seem that the majority probably did not have ulcer. Osler and McCrae, as stated above, found about the same percentage of mild or vague gastric complaints among a series of hospital cases as among their gastric carcinoma cases. Certainly it is true that a very definite percentage of hospital patients will give a history of some gastro-intestinal disturbance.

There were eleven cases (11.3 per cent.) in the series of gastric malignancy that had a duration of five years or over. This included the five cases just mentioned. On the basis of duration of symptoms alone it would appear that 10 per cent. would be a high value for incidence of carcinoma in ulcer of the stomach.

SEX DISTRIBUTION

Three-quarters of the cases of ulcer occurred in the male as against 86 per cent. in the carcinoma series. There is thus seen to be no real difference in the two conditions in so far as sex distribution is concerned.

APPETITE

Slightly over one-half (58.6 per cent.) of the twenty-nine cases of ulcer in which a state-

ment was made relative to the appetite had such recorded as "good or better" as against about one-quarter (26.7 per cent.) of the cases of carcinoma of the stomach. In other words, a good appetite is about twice as common in subjects with ulcer of the stomach as with those with cancer.

LOSS OF WEIGHT

In reviewing the cases in this series with this topic in mind, it seemed that the rate of loss of weight was more suggestive than the actual amount lost. It was found that about one-sixth (17.6 per cent.) of thirty-four cases of ulcer showed a loss in weight of twelve pounds or more in six months as compared with about one-half (50.7 per cent.) of seventy-three cases of carcinoma of the stomach. There is then a much more rapid loss of weight in gastric malignancy than in gastric ulcer.

GASTRIC ANALYSIS

The main point of difference between the two conditions shown by this test was the amount of free hydrochloric acid in the gastric contents. Thirty of the gastric ulcer cases and forty-nine of the subjects with carcinoma of the stomach were available for this comparison.

Free hydrochloric acid was absent in the fasting contents in approximately one-third (30 per cent.) of the gastric ulcer and nine-tenths (87.7 per cent.) of the cancer cases. Only one-tenth of the cases with gastric ulcer showed absence of free hydrochloric acid in contents removed after test meal as compared with about three-quarters (70.8 per cent.) of the cases of gastric malignancy. The absence of free hydrochloric acid in the gastric contents, therefore, seems to be much more common in cancer than in ulcer of the stomach.

STOOLS

The finding of note in this connection seemed to relate to the presence or absence of occult blood. Forty-eight cases of carcinoma and thirty cases of ulcer were used for this comparison. The guaiac test was positive in about three-quarters (70.6 per cent.) of the cancer cases and in only about one-quarter of the ulcer cases. In other words, occult blood is found about three times as often in the feces of patients with carcinoma of the stomach as in patients with ulcer.

BLOOD

There was no difference of note in the two series of cases as regards red blood cells or hemoglobin.

PHYSICAL EXAMINATION

The one outstanding physical finding differentiating these conditions was found to be the presence or absence of a palpable abdominal mass. Over one-half (52 per cent.) of the cases with carcinoma had a palpable mass whereas there was only one of the cases of ulcer that gave this finding.

Emaciation, of course, is much more common in carcinoma than ulcer.

INITIAL SYMPTOM AND CHIEF COMPLAINT

The most common initial symptom and chief complaint in both sets of cases was epigastric pain. The cases of carcinoma giving a history of two years or more in duration were separated to see if there were any initial symptoms which would be particularly helpful in the early diagnosis of this condition. A review of these cases has shown that pain is the most common initial symptom. However, "gas" was found at the onset so frequently as to make it seem worth while to sound a warning relative to the presence of this symptom in those beyond the third decade. Heart-burn and recent onset of constipation are other gastro-intestinal symptoms that should not be overlooked in subjects in the cancer age. It seems logical that any adult subject with unexplained gas on the stomach, heart-burn or constipation should have a gastro-intestinal X-ray at once.

CONCLUSION

Certain very definite differences are to be found in the clinical finding of ulcer of the stomach and gastric malignancy. These seem to indicate that there is not a very close relationship between the two conditions. Ten per cent. would seem to be a very high value to give to the cases of ulcer of the stomach which later become malignant.

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TOURNAMENT WINNERS

THE reduction in the number of National Tournaments in health knighthood from two to one does not seem to have affected the enthusiasm of Crusaders the country over.

Nearly four thousand schools have been awarded pennants for the successful completion of twelve consecutive weeks of faithful performance of the Crusade health rules during the school year just past.

THE DIAGNOSTIC SIGNIFICANCE OF LATERAL CURVATURE OF THE SPINE CAUSED BY MUSCULAR SPASM

BY JOHN RUSSELL CARTY, M.D.

It is well known that reflex spasm of certain groups of spinal muscles may give rise to lateral curvature. During the past two years the writer has been impressed with the diagnostic value of such a deformity in obscure pleural and renal pathological conditions. The purpose of this article is to discuss briefly this type of scoliosis and to evaluate the diagnostic significance.

The spine as a whole can be likened to a flexible column. This column is supported by mutually antagonistic muscle groups acting against gravity. The resultants of such forces keep the spine in perfect equilibrium and give to it the familiar contour. Any over-action of a muscle group, as opposed to the antagonistic muscles, tends to destroy the normal balance and in many cases will produce either abnormal curves or exaggeration of the normal ones. One of the most common deformities so produced is a lateral curve, which we will designate as a spasmodic scoliosis. Spasm of the spinal muscles may also cause an exaggeration or diminution of the normal anteroposterior curves. The observations which form the basis of this article have been made on the lateral curvatures only.

Excluding lateral curvatures following pathological processes or congenital anomalies of the vertebrae themselves, scoliosis caused by spasm may be confused with the so-called functional or postural type. The deformity here seen consists usually of a large or so-called main curve with secondary deformities pointing in the opposite direction above or below. Occasionally only one curve is seen. In general the deformity is extensive and involves more than one region of the spine. There is usually some rotation of the bodies and, if the deformity is severe and long standing, there are changes in the shape of the vertebrae.

The spasmodic curve is generally single and, what is most important, *the convexity points away from the affected side*. The deformity is usually local and limited to one region. If, however, the spasm is of long standing, there is a readjustment of equilibrium in the entire spine, and then the deformity may be quite extensive. Rotation of the bodies is comparatively rare except in cases of very intense spasm, such as seen after trauma to the muscles. There is no alteration in the shape of the vertebrae. The frequency and the extent of the deformity depends to a certain degree on the suppleness of the spinal column. Consequently, we see the deformity more frequently and more marked in children.

The smaller degrees of spasmodic scolioses are often overlooked during a general physical examination. The deformity is slight and there may be no symptoms referable to the spine. Radiographically the deformity is quite apparent. Care must be taken, however, to eliminate error which might be caused by faulty position while taking the radiograph. This is particularly true in the case of children, as they are very prone to slump.

A spasmodic scoliosis may be associated with the following conditions: (1) trauma to the spinal muscles, (2) infection of the spinal muscles, (3) pleural effusion, (4) renal stone, (5) hydronephrosis, (6) perinephritic abscess. Pathological processes of the vertebrae or intervertebral spaces and congenital anomalies are not included, as they are usually obvious, and the presence of a lateral deformity would be of little help in the diagnosis of such conditions.

Very marked lateral deformity is often seen following trauma to the spinal muscles, such as tearing loose of muscle attachments. Scoliosis is more noticeable if the trauma is confined chiefly to one side. The spasm may be so intense as to cause rotation of the bodies.

Illustrative case: The patient, a heavy-set white woman, forty-eight years old, fell down stairs several hours before coming to the clinic. In attempting to arrest her flight down the stairs she "felt something give way" in her right groin, followed immediately by severe pain and stiffness. Physical examination showed marked lumbar scoliosis with the convexity pointing to the left. There was marked spasm in the right lumbar region. Radiographic examination confirmed the physical findings. No fracture was seen.

Myositis of the spinal muscles, particularly if unilateral, may be accompanied by marked spasm. A familiar example of this is the deformity of wry neck following myositis of the sternocleidomastoid muscle.

While making an extensive study of the radiographic aspects of empyema in children, the writer noted that thoracic scoliosis was seen in 78% of the series of pleural effusion. This group included both purulent and nonpurulent effusions. In a corresponding series of cases of pneumonia uncomplicated with fluid in the pleural cavity the deformity occurred in a negligible percentage. Most of the cases not showing lateral deformity were those in which the effusion was large and there was displacement of the heart. It is felt that in the differential diagnosis between pneumonia and purulent pleural effusion in children the absence of this sign is strong evidence against the existence of fluid in the pleural cavity.

Subsequently a similar study was undertaken in adults. As would be expected, a smaller percentage of pleuritic effusions showed a lateral

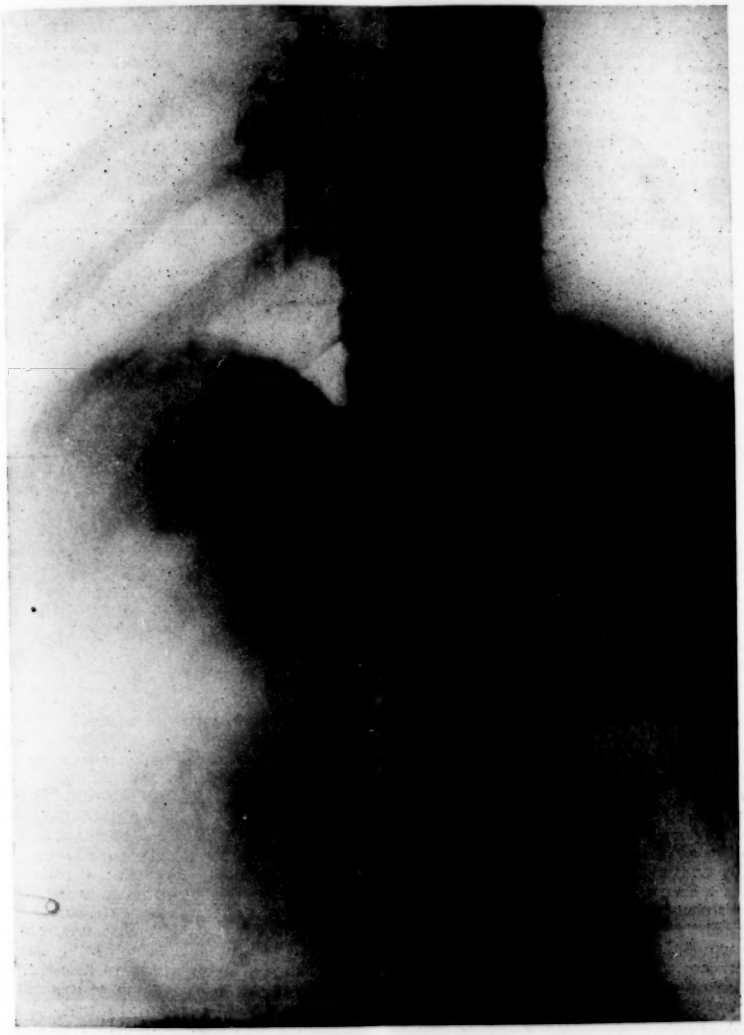

ILLUSTRATION NO. 1. A spasmodic lumbodorsal scoliosis to the left. Note the large calculus on the right of the first lumbar vertebra. This stone was "silent."

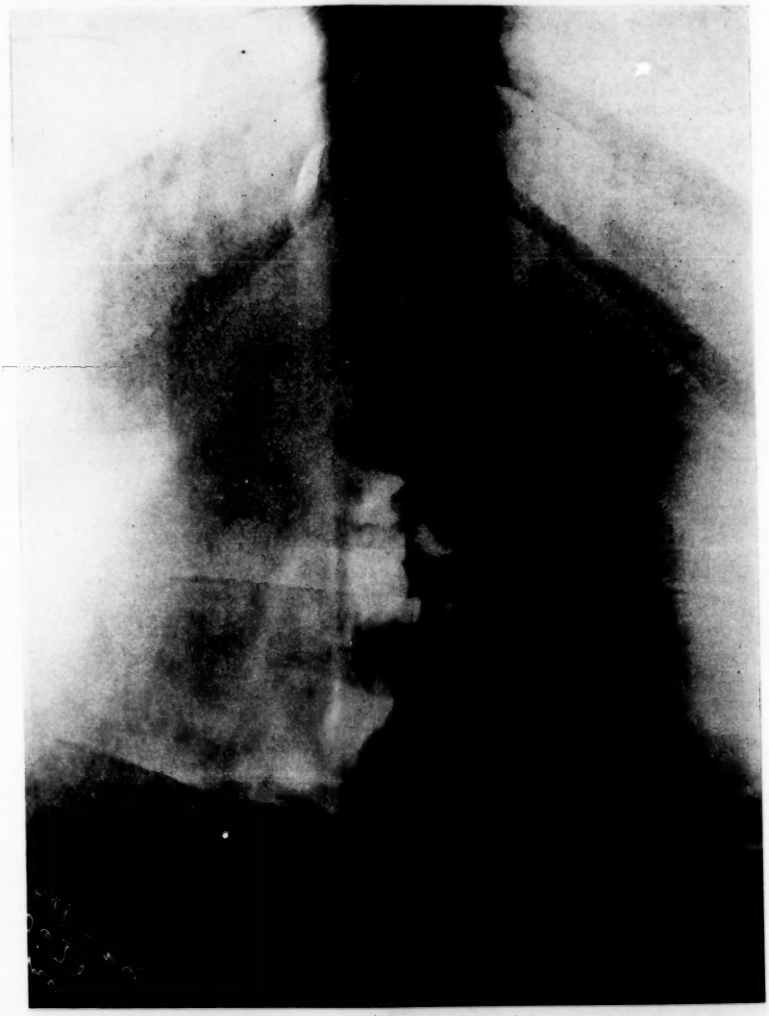

ILLUSTRATION NO. 2. A marked lateral spasmodic curve caused by muscular trauma. The convexity points away from the injured side.

curvature. Almost half of the series collected of 200 cases showed the deformity. Obviously, the absence of scoliosis would have no diagnostic value. It is an interesting fact that in both series no curve was noted in which the convexity pointed towards the lesion. It should be distinctly understood that the scoliosis referred to occurs early in the disease and is entirely distinct from the later deformities seen in chronic empyema.

Illustrative case: The patient, a well-nourished male child of six years, had a sudden attack of coughing associated with fever five days before coming to the hospital. Since then he has had an irregular fever with cough. Physical examination showed some impairment of resonance of the lower half of the left chest with faint breath sounds and no definite bronchial breathing. An occasional moist rale was heard over the upper left chest. Physical examination of the remainder of the chest was negative except for scattered moist rales on the right. Radiographic study showed an area of moderately increased density occupying chiefly the lower half of the left chest. The costophrenic angles were clear. The heart was not displaced. There was a moderate thoracic scoliosis, the convexity of which pointed to the right. On the basis of the deformity, fluid in the pleural cavity was strongly suggested, which was demonstrated only after repeated needling of the chest.

Following the observations made in the thoracic region, attention was directed to possible lateral deformities in pathological renal conditions. The deformity was quite constant in acute attacks of renal colic. In this case, however, the clinical picture is usually clear-cut. The deformity was found in approximately 60% of 100 cases with large renal stone. Inasmuch as this type of calculus may give rise to little or no pain or other symptoms, the presence of a lateral deformity may be of considerable importance, as shown by the following case:

The patient, an undernourished white married woman of forty-three years, came to the clinic complaining of attacks of occipital headache associated with vomiting and dull pain in the right lower abdominal quadrant. The symptoms began about six months previously. Her past history was negative except for an appendectomy eight years previously. The physical examination was essentially negative, except that reduplication of the apical second heart sound with a presystolic murmur at the base was noted. There was an appendectomy scar in the right lower quadrant. The urine examination was negative. The patient was sent to the X-ray Department for radiographic study of the heart. A moderate lumbodorsal scoliosis to the left was noted. The possibility of a renal stone was suggested. A radiograph of the genitourinary tract showed a large, dense stone in the pelvis of the right kidney. Pyelographic study revealed a hydronephritic kidney on this side.

Hydronephrosis, particularly if extensive, may give reflex deformity. Obliteration of the kidney outline, together with a lumbar scoliosis whose convexity points away from the suspected side, should suggest the possibility of a hydronephrosis.

Illustrative case: The patient, a pale, young, single man of twenty years, came to the clinic complaining of dull pain in the left lower quadrant, which began eighteen months previously. There was hematuria at the onset of the pain and he has had nocturia since then. Physical examination showed nothing of interest. The urine contained an occasional white blood cell but no definite pus. A radiograph of the genitourinary tract showed an obliteration of the kidney outline on the left side with spasmodic scoliosis, the convexity of which pointed to the right. Pyelography was advised, the presence of a hydronephrosis being strongly suspected. A huge hydronephrosis was demonstrated.

Several cases of perinephritic abscess were observed with lateral lumbar deformity. The deformity, however, was not marked.

Conclusions: First, attention is called to the fact that a lateral curvature of the spine caused by spasm may have considerable diagnostic significance. Second, the presence or absence of thoracic scoliosis in obscure chest conditions in children is of considerable diagnostic value. The presence of this deformity is of value under the same condition in adults. Third, lumbar scoliosis may direct attention to a renal stone, hydronephrosis, or perinephritic abscess, possibly not suspected.

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DEHISCENCE OF THE FLOOR OF THE MIDDLE EAR*

BY DANA W. DRURY, M.D., F.A.C.S.

THE principal point in connection with the surgical anatomy of the tympanum is its relation to other parts. Its roof is formed by a thin plate of bone, which, with the dura mater, is all that separates it from the temporosphenoidal lobe of the brain. Its floor is immediately above the jugular fossa behind and the carotid canal in front. Its posterior wall presents the openings of the mastoid cells. On its anterior wall is the opening of the Eustachian tube. Thus it follows that in disease of the middle ear we may get subdural abscess, septic meningitis, or abscess of the cerebrum or cerebellum from extension of the inflammation through the bony roof: thrombosis of the lateral sinus, with or without pyaemia, by extension through the floor: or mastoid abscess by extension backward. In addition to this, we may get fatal haemorrhage from the internal carotid in destructive changes of the middle ear: and in throat disease we may get the in-

*Contributions from the Evans Memorial, No. 85, A-41.

flammation extending up the Eustachian tube to the middle ear.

Anatomically, the inferior wall is narrower than the superior; it is bounded behind by the posterior wall, and in front by the gentle elevation of the inferior towards the anterior wall, which is situated below the tympanic orifice of the Eustachian tube. The position and form of the floor of the tympanum depend on the size of the fossa jugularis, the latter being deeper on the right than on the left side. On its upper surface it is provided with narrow bony crests which, together with the entire osseous layer, represent the rudiments of the bulbus tympani. The latter is very strongly developed in some animals, and can still be demonstrated in the monkey. The thickness of the osseous layer varies considerably, being sometimes as thick as five to eight mm., while in other cases it is as thin as paper and even sometimes punctured by small holes. In rarer cases it is entirely absent, so that the fundus of the tympanic cavity merely consists of connective tissue, and the bulb of the jugular vein protrudes into the tympanic cavity. If the floor is very thick it contains air cells, and if it is thin, it usually consists of a compact bony layer which does not contain very large areolae. In several specimens the dehiscence has been so extensive that the jugular fossa has even extended to the internal auditory meatus, so that the internal auditory canal will communicate with it by an aperture.

As the literature on this subject is rather small, and the cases cited as references only, it has seemed useful to collect the material of the case histories of those cases that have been reported; naturally they fall into one group, namely operative cases. To this summary of the literature we add a single case that came under our personal observation.

Toynbee, in his extensive collection of anatomical specimens, describes no case reports but several of the specimens as follows:

TABLE I

Reporter	Year	No. of cases	Sex	Age	Disease	Operation	Treatment	Remarks
Brieger	1854	1	M	56	Roma	Inc. M. T.	Tamponage	Sinus thrombosis: Death
Zaupal	1867	1	Inf.	—	Roma	Rupt.	"	Recovery
Gogefroi	1881	1	M	8	Roma	Inc.	"	Sinus thrombosis: Death
Hildebrandt	1890	1	F	4	Oma	"	"	Recovery
Ludewig	1890	1	M	5	Roma	"	"	"
Gruber	1891	1	M	41	Oma	"	"	"
Seligman	1893	1	M	—	Roma	"	"	"
Gomperz	1894	4	M	30	Roma	"	"	"
			M	25	Loma	"	"	"
			—	—	Ch. oms	"	"	"
			F	13	Roma	"	"	"
Dench	1898	1	F	—	Ch. oms	—	—	Ossiculectomy; ligation jugular; recovery
McKernon	1898	1	F	13	Oma	Inc.	Tamp.	Arterial bleeding; recovery
Von Rohre	1901	1	M	7	Oma	"	"	Recovery
Compaireo	1902	1	—	—	Oma	"	"	"
Max	1906	1	—	4	Roma	"	"	"
Hassblauer	1911	1	—	—	Roma	"	"	"
Lüders	1912	2	F	42	Oma	"	"	"
			—	Inf.	Oma	"	"	Mastoid oper.; thrombosis: Death
Goerke	1914	1	—	—	Oma	"	"	Mastoid oper.; recovery
Page	1914	1	—	Inf.	Boma	"	L. Tamp.	" " "
O'Connell	1907	1	M	23	Roma	"	"	Recovery
Shapleigh	1918	1	—	—	Roma	"	"	"
Total: 19	1854-1925	23	M=10 F = 4 Inf. — 56		Roma=12 Loma= 2 Ch. oms=2	Inc.=21 Rupt.=1		Sinus Thrombosis=4 Ligation Int. Jugular=1 Mastoid Oper.=4 Deaths=3 Recoveries=20

No. I—Man, age 54 years, whose membrana tympani was flatter and much thicker than usual; the lower half being white, like parchment; the mucous membrane lining the tympanum was thick, especially the portion surrounding the ossicles; the layer of bone between the jugular vein and the tympanum was incomplete. The stapes was fixed more firmly in the fenestra ovalis than is normally found.

No. II—From a man, age 60. The lower wall of the tympanum was formed by a membrane, in which a small plate of bone was deposited. The membrana tympanum was very concave.

No. III—The lower wall of the tympanum formed partly by a very thin and translucent plate of bone, and partly by a membrane. It presented an orifice of oval shape, about a line in length.

No. IV—Lower wall of tympanum very thin, and presents an irregular triangular orifice, about a line and a half in diameter.

No. V—Lower portion of lower wall of tympanum consists of membrane only.

No. VI—Lower wall of tympanum very thin and translucent; for a space about the size of small pea it consists of a layer of membrane, containing a little osseous deposit.

No. VII—Lower wall of tympanum very thin and translucent, and perforated by many minute orifices.

No. VIII—Lower wall of tympanum translucent, and presenting a small oval orifice.

No. IX—Orifice in lower wall of tympanum with two or three minute orifices around it. Lower wall of meatus externus also presents an orifice.

No. X—Larger portion of lower wall of tympanum is deficient.

No. XI—Lower wall of tympanum is entirely absent, except a very thin lamina of bone at the inner angle.

No. XII-XIV—Lower wall of tympanum is perfect, but very thin and translucent.

No. XV-XVI—Lower walls of tympanum imperfect in several places presenting several minute orifices.

No. XVII-XXII—Lower wall of tympanum consists in part of membrane only.

No. XXIII-XXVI—Lower wall of tympanum deficient in parts.

No. XXVII—Orifice in the layer of bone between the lateral sinus and the mastoid cells, which allows communication between the two cavities.

Hartmann's collection of one hundred temporal bones yielded five with the floor of the tympanum thin as paper, two bones showing dehiscences, while a third specimen of the group showed very small perforations in the floor of the tympanum. Zuckerhandl, on the other hand, found great enlargement of the fossa jugularis in nineteen of sixty-seven specimens, or 35.2%. Korner examined four hundred and forty-nine temporal bones and found twenty-three cases of pin hole perforations to multiple perforations in the floor of the middle ear, or in 19.5%. Zuckerhandl mentions the fact that the vein may be in direct contact with the interior of the tympanic cavity, with the optic and acoustic nerves and even reaching the superior petrosal sinus. Barth, in reporting his anatomical work speaks of dolichocephalic and brachycephalic skulls, that in the former type, the bony floor of the tympanic cavity is generally 1 mm. thick, containing often air spaces, while in the latter type, the bony floor is thinner, and often transparent. In the case of dehiscence, not one instance in thirty-nine dolichocephalies was found, but of ninety-two brachycephalies nine instances were noted, and in one of the latter skully the abnormality was found as bilateral.

Most of the text books mention the condition as taking place, especially in the anatomical sections, but very few actual cases in life are even mentioned. There seems to be some confusion also, whether the condition was brought about through suppuration or whether it might not be a congenital defect. Again, it has been rather difficult with some of the case histories to determine whether the bleeding was arterial or venous in origin.

The present case is reported as a matter of record:

Wm. B., six years old, had always been well and active until exposed to scarlet fever, which he developed in a mild form. In the second week of the disease, early one morning, he became more fretful and his temperature rose to 102F. The baby was quiet if left on the right side and the right ear nestled into the pillow. On disturbing, the child would cry and show signs of pain. The ears were examined and while the findings in the left ear were not remarkable the right canal was abnormally red and the drum membrane diffusely injected and bulging superiorly and posteriorly. The nose, throat and residual physical examination showed nothing of note. The child was given primary ether, and, following sterilizing of the canal with an alcohol wick, the drum was incised from above downward. Before the knife could be withdrawn from the ear canal the entire area seemed to be filled with blood extending out onto the face. Light packing of the canal failed to control the bleeding. A longer, larger piece saturated in adrenalin was placed firmly into the canal. A pad was quickly placed over the ear and a tight bandage rolled about the head. This controlled the bleeding, although a small amount of blood ran into the nasal chambers and a small amount also could be seen in the pharynx. This event taking place in the morning, the pad was removed in the evening, as the child was very uncomfortable. Later in the evening the plug in the canal was loosened slightly, but as the bleeding began afresh (although less actively), a clean pack was reinserted together with a pad and bandage. The next day there being no further signs of bleeding an attempt was made to remove the plug, but again the bleeding became active, and the same procedure was carried out again. Meanwhile the clotting time was taken and a cover slip of the blood was examined, but nothing abnormal was found. On the evening of the second day the plug was removed without further bleeding except the serum discharge from the middle ear. The temperature being normal and the condition now giving the appearance of a serous middle ear, a sterile wick was gently placed in the ear canal, this being changed as often as it became saturated with serum. During this episode the left ear quieted and became normal in two days. The right ear became dry in eleven days following the incision. On the fourth day of the illness a white count showed no active blood stream infection. Six weeks following the acute otitis I had another opportunity of observing the ear drum and it showed a healed membrane the inferior margin of which looked darker. On wiping over the drum this darker area was very evident, although no pulsation could be made out. The short process of the drum seemed to be more prominent than usual.

In the discussion of this type of case as occurring in practise it seems wise perhaps to mention certain points that I have found emphasized by a number of authors while looking over the literature. Trautmann points out the anatomical peculiarities of certain temporal bones which he called dangerous. In these the sigmoid sinus runs far forward, curves inward, and passes over the projecting ledge which forms the posterior boundary of the jugular foramen. Then the jugular bulb rises against the floor of the tympanic cavity and lies on a higher plane than the lowest point of the sigmoid sinus. Politzer mentions the possibility of the fascial nerve being exposed in cases of a dehiscence and in such a case a phlebitis happened followed by a paralysis of the face. Hildebrandt, on the other hand, calls attention to the observation that on closely observing the drum membrane in such cases the bulging changes its shape, and that at one time it will be higher than at another time, while Gruber mentions a change in the shape of the cone of light by pressing the jug-

ular on the side of the neck. When this happened he concluded that there was a dehiscence and would not incise the drum membrane. Ludewig and Seligman both mention the fact that the drum membrane it much more resistant to the knife, perhaps not so much the drum membrane but the wall of the vessel behind it that gives the sensation of resistance. This point I recall distinctly as I opened the drum membrane in the case cited above. Haemophilia is raised by several authors but in none of the actual cases did this seem to be the cause. McKernon's case showed the bleeding to be synchronous with the pulsations of the radial artery and further that it was bright red in color; he held in his case that it was probably a rupture of one of the blood vessels that supply the tympanic cavity. Korner raises the point of a phlebitis following in this type of case. This fact did happen in the first case that I reported which extended to the meninges along the jugular vein. Eitleberg thinks that there is a possibility of death from haemorrhage in these cases, although I have failed to find any so recorded. Bertemes suggests that the mucous membrane and the vessel wall being in apposition there is potentially a hernia at this point into the tympanic cavity, thus it is relatively easy for the infection to enter directly into the blood stream: This point may account for some infections of the middle ear causing phlebitis, although I have never seen this mentioned as a location of a thrombosis as a primary location. Some authors refuse to do an incision of the drum membrane when the posterior quadrant shows a blue color, but I can see no objections of incising the membrane higher and in the anterior area. Max mentions the fact that with the Siegle's otoscope, the tympanic membrane lifts and the color changes to a grey white and that the color is less intense on the normal side. These two points are confirmed by Urbantschitsch, while Politzer states that he has seen cases with the same blue coloring and where the bulb was still separated from the cavity by an osseous lamella. Gomper, on the other hand, could see no difference by compressing the jugular in the neck. Personally I believe that in an inflammed membrane any change in color through compression on the neck is a negligible quantity as since I have had the experience of the case mentioned above I have tried this test repeatedly and have failed to satisfy myself that it is a practical test except in a serous ear wherein small changes of color are susceptible of reasonable interpretation. McKernon, in 1905, called attention to the fact that in children there is anatomically a closer relationship between the floor of the tympanum and the bulb. Schwartz must have had the danger in mind when incising the drum as he never would allow the drum to be incised from above downward, but always from below upward. Rohre mentions the observation that the external veins of the face on the same side are usually more full and seem to be larger than normal, especially the external jugular.

CONCLUSIONS.

1—It is difficult to determine whether the bleeding is venous or arterial.

2—Proper tamponage of the external ear will control any haemorrhage that can occur from the middle ear.

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BENIGN GLYCOSURIA WITH HYPERGLYCEMIA: REPORT OF CASE WITH METABOLIC STUDIES*

BY ELOISE PARSONS, M.D.

THE case presented here has many interesting features. A young woman had mild diabetes for nine years at an age when diabetes is likely to be severe and progressive. Glycosuria was discovered while she was pregnant. The degree of diabetes has not changed during the four years she has been under observation. Hyperglycemia and glycosuria appear after the ingestion of glucose. On a restricted carbohydrate diet there is always glycosuria.

A few cases of somewhat similar features have been reported. They fall into a general category of diabetes innocens, a condition

*Submitted for publication June 1, 1926.

which has been discussed by Frank, Salomon, Riesman, Campbell and others.

Woodyatt recently presented a case of mild diabetes in an elderly person with arteriosclerosis, and illustrated a method of study by which the utilization of glucose was estimated. The diet containing a definite supply of glucose was increased by a certain amount of carbohydrate at intervals of three days during which the twenty-four hour excretion of sugar in the urine was estimated. The difference between the supply of glucose and the excretion determine the utilization of carbohydrate by the body.

Wilder and associates have studied the utilization of carbohydrate by determining simultaneously the respiratory exchange, the blood sugar and the urinary excretion. Recently Ladd and Richardson, and Finley and Rabinowitch have applied this method in the study of renal diabetes. In the case presented here metabolic studies were made by both methods.

REPORT OF CASE

A young woman, aged twenty-eight, came to the clinic in January, 1922, because of threatened abortion. The symptoms were abdominal cramps, nausea and vomiting, and vaginal bleeding. She also had diabetes. There was no history of diabetes in the family. Glycosuria had been discovered in the routine urine examination during her first pregnancy in 1917. She was told that it would disappear after delivery. The pregnancy terminated normally. She nursed the baby and was fairly well, although underweight.

Examination of the urine in the summer of 1919 did not disclose sugar. The patient consulted a physician because of fainting attacks which began with headaches followed by marked weakness and trembling. In November, 1920, she consulted a physician because of weakness and loss of energy. Excessive amounts of sugar were present in the urine. At this time there was also a slightly increased thirst, increased frequency of urination, and a peculiar craving for potatoes, although these symptoms were not prominent. Even after the patient was put on a restricted carbohydrate diet, sugar continued to be excreted. Gradually all food was eliminated from the diet except toast and postum. She lost weight and was very weak.

In February, 1921, the patient was on a milk diet for two weeks. Because glycosuria persisted starvation was resorted to for five days, but the urine did not become quite sugar-free. Three severe attacks occurred during the starvation period when she became stiff and cold; each attack lasted from thirty to forty minutes. A diet with restricted carbohydrates was prescribed. Examination of the urine from time to time over a period of nine months disclosed traces of sugar, usually less than 1 per cent. In December, 1921, the patient became pregnant again and came to the clinic two months later.

Examination.—On admission the patient appeared to be undernourished. She was 5 feet, 4 inches tall and weighed 107 pounds. Besides the pregnancy the only significant physical findings were the emaciation and a small soft colloid goiter.

The hemoglobin was 67 per cent, erythrocytes numbered 3,700,000; the leukocytes 7,000. The blood sugar was 100 mg. for each 100 c.c. and the urine contained 3.12 gm. of sugar in a twelve-hour specimen. The systolic blood pressure was 115, the diastolic 80. As abortion seemed inevitable, the uterus was emptied under nitrous oxid and oxygen anesthesia. The patient recovered promptly. She was transferred to the diabetic service and has been under observation since.

Table 1

Changes in blood sugar in case of benign diabetes

Date	Blood sugar, fasting, mg.	Glucose ingested, gm.	Blood sugar after ingestion of glucose, mg.				
			Hours				
			0.5	1.0	1.5	2.0	3.0
2-26-22	0.097	69	0.333			0.200	0.109
2-21-23	0.095	50	0.333			0.154	0.090
3- 7-26	0.085	50	0.190			0.180	0.115
3-10-26	0.095	50	0.148	0.202	0.179	0.175	0.113

CLINICAL OBSERVATIONS DURING FOUR YEARS' DIABETIC MANAGEMENT

In the hospital the urine became sugar-free when the patient was on a diet consisting of 60 gm. carbohydrate, 60 gm. protein and 125 gm. fat, but sugar appeared when the carbohydrate allowance was raised 10 gm. The blood sugar was normal. The blood fats, and the acetone and carbon dioxid-combining power were normal. The basal metabolic rate was −1.

The patient went home on the foregoing diet after having been well trained to prepare a weighed diet and to carry out instructions. After two weeks she wrote "Can I increase my diet? I find that it is impossible to do much work on the present number of calories." The diet was increased by 10 gm. of protein and 25 gm. of fat. A month later she wrote "I find the new diet very satisfactory but must admit that I am still hungry." In August, about six months after her examination in the clinic, she had gained to 115 pounds, but the diet seemed insufficient. Traces of sugar appeared in the urine occasionally.

In February, 1923 the patient returned to the hospital for a check-up examination. On the diet prescribed she showed traces of sugar in the twenty-four hour specimen of urine. The test for sugar tolerance showed the blood sugar to be 333 mg. for each 100 c.c. one-half hour after the ingestion of 50 gm. of glucose. She seemed in such good physical condition, that she was advised to continue the diet, although she complained of being hungry.

She continued on the same weighed diet until she again returned to the hospital in February, 1926. At this time she weighed 117 pounds and appeared well nourished although her weight was 20 pounds below the standard for her height and age, and 15 pounds below her maximal weight ten years previously. She

reported that there are always traces of sugar in the urine.

The blood sugar response to glucose test meals was obtained in 1922, 1923 and 1926 (Table 1). The curve in every instance differs from that usual in metabolic tests, each over a fifteen-minute period, and blood sugar and urine estimations were made over a period of seven hours.

In this case the respiratory quotient shows the characteristic rise which occurs in a normal person

Table 2

Effect of ingested glucose on the respiratory exchange, urinary excretion and blood sugar in case of benign diabetes

Period	Respiratory exchange.			Urinary excretion, gm.			Blood Sugar
	Total calories	Respiratory quotient	Metabolic rate	Amount	Specific gravity	Sugar	
Fasting	56.6	0.76	-3	25	1.012	0	0.095
	55.6	0.77	-1				
Ingestion of 100 gm. glucose in 500 c.c. water, juice one lemon, March 20, 1926							
One-half hour later	60.8	0.90	+6	500	1.006	17.5	0.199
One hour later	64.1	0.90	+12	325	1.010	7.5	0.207
Two hours later	65.3	0.93	+14	135	1.020	1.4	0.159
Three hours later	60.7	0.78	+6	50	1.007	0.4	0.036
Five hours later	61.1	0.77	+7	125	1.020	0.5	0.067
Seven hours later	63.8	0.76	+11	275	1.004	0	0.098

Table 3

Metabolic findings in case of benign diabetes

Date, 1926	Weight, pounds	Diet					Urine					Blood sugar, gm. for each 100 c.c.	Summary		
		Carbohydrates, gm.	Protein, gm.	Fat, gm.	Calories	Metabolic glucose, gm.	Volume, c.c.	Glucose		Nitrogen	N x 6.25		Carbohydrate intake, gm.	Carbohydrate output, gm.	Carbohydrate excreted, per cent
								Grams	Average, gm.						
3-7	117	60	70	180	2207	119	875	6.1				0.94			
3-8	117	60	70	180	2207	119	1225								
3-9		60	70	180	2207	119	1450		6.1				119	6.1	5.1
3-10	Glucose test-meal														
3-11	115	90	70	180	2260	149	1575					0.91			
3-12	115	90	70	180	2260	149	1350	8.1		6.3					
3-13	115	90	70	180	2260	149									
3-14	115	90	70	180	2260	149	1850	11.0	9.5	7.4	42.5		149	9.5	6.4
3-15	117	120	70	180	2380	179	1925	10.5		4.7		0.95			
3-16		120	70	180	2380	179	1850	27.0*		9.4					
3-17		120	70	180	2380	179	1650	8.5		7.2					
3-18		120	70	180	2380	179	1250	5.6	11.5#	6.0	44.4		179	11.5	6.4
3-19	118	150	70	180	2500	209	1650	16.3		7.1		0.92			
3-20		150	70	180	2500	209	1900	12.3		6.5					
3-21		150	70	180	2500	209	1250	12.5	13.8	5.5	39.4		209	13.8	6.6
3-22	119	180	70	180	2620	239	1750	10.7		6.1		0.99			
3-23		180	70	180	2620	239	2000	9.0		6.9					
3-24		180	70	180	2620	239	1600	9.3	9.7	6.5	40.8		239	9.7	4.05

* Acute upper respiratory infection.
# Average of three days. • Not included.

cases of diabetes in that it returns to normal at the end of three hours.

The day before the experiment (Table 2) the patient was on a basal diet and in bed. The following morning the metabolic rate during fasting was obtained. Blood was drawn and urine collected for the purpose of estimating the sugar. Lemonade containing 100 gm. of glucose was ingested. Subsequent after glucose is taken. The metabolic rate also shows a normal increase.

Large amounts of sugar were excreted in the urine, 17.5 gm. in the first half hour. The blood sugar curve rises much too high to be normal, but returns to a low value in the time considered normal. The most interesting feature in this table is the extremely low level to which the blood sugar fell following the

hyperglycemia. During the two hours when the blood sugar was 0.036 and 0.067, the patient was in distress. She complained of headache and hunger. She was quite restless, although ordinarily very composed. A tremor of the hands and perspiration on the brow were noted. The patient says that she has the same sensations in less degree when she suffers from hunger between meals.

The metabolic findings for two weeks are given in detail in Table 3.

As the diet was increased in carbohydrate, the protein and fat were kept constant, so the only variable is the glucose intake. This was increased 30 gm. every three or four days. The excretion of sugar in the urine did not increase in the same proportion, although it was always excessive. The percentage of the sugar excreted remained nearly constant. The blood sugar before breakfast remained constant.

There were no symptoms of diabetes at any time, no polyuria, no thirst. The patient stated that when she was on the high-carbohydrate diet, she was better than she had been for years. For the first time since she began dieting, she was not hungry. The gain in weight is noticeable.

COMMENT

No attempt is made to classify the type of diabetes present in this case. It is not the so-called renal diabetes because the blood sugar rises too high. After glucose test-meals, carbohydrate is utilized in a normal manner as shown by the rise in the respiratory quotient, yet at the same time the blood sugar is abnormally high. The metabolic findings, using Woodyatt's method of study, parallel those he reported in a case of mild diabetes.

The onset during pregnancy adds to the perplexity of the case since it places it in the confusing group of pregnancy glycosuria, some of which become very severe, while others disappear with the termination of the pregnancy.

The very low blood sugar, 0.036 gm., observed five hours after the glucose meal of March 26, 1926 was attended with symptoms of hypoglycemia. Such reactions have occasionally been observed in normal persons after glucose test-meals especially when the carbohydrates in the diet of the preceding twenty-four hours have been rigidly restricted. The symptoms attending the reactions are identical with those observed after an overdose of insulin.

In retrospect it appears that this patient was subjected to unnecessary dietary restriction. It would not have been justifiable, however, to have treated her otherwise. To conclude that the case was benign would not have been warranted within a shorter period of observation. The harm done by insufficient treatment in a case of true diabetes is far greater than that attending somewhat excessive treatment in the relatively infrequent cases of diabetes innocens.

CONCLUSION

Benign glycosuria was diagnosed in this case, and a good prognosis offered because of the following facts:

1. Glycosuria is of long duration and it has persisted nine years without increase in severity.
2. Sugar is excreted on restricted diets and its excretion on unrestricted diets is no greater in proportion to the intake of carbohydrates.
3. The blood sugar remains normal even when the diet is rich in carbohydrates, as tested by a sample obtained before breakfast.
4. The respiratory quotient responds to a glucose meal as readily as in normal persons.

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AN UNUSUAL BLOOD SUGAR FINDING DURING AN INSULIN REACTION*

BY MILLARD SMITH, M.D.

THE following insulin reaction is reported because the unusual blood sugar finding may have some significance in the question concerned with the nature of the reducing substances which react with the blood sugar reagents used in the method of Folin and Wu[1].

Benedict[2] has introduced a new method for the determination of blood sugar which gives lower values than that of Folin and Wu. He obtained blood sugar values, in the blood obtained from a dog in convulsions due to a large dose of insulin, of 27.0 mgs. and 16.0 mgs. by the methods of Folin and Wu and his own respectively. He states that he believes the blood was actually free from glucose and that even his method gave too high a result.

More recently Folin[3] has introduced a modification of the original Folin-Wu blood sugar reagents and obtains values even lower than by the method of Benedict. These workers seem to believe that the present blood sugar methods determine glucose plus an X-reducing substance.

*From the Thorndike Memorial Laboratory, Boston City Hospital.

Therefore even though insulin may reduce the blood glucose to zero, by their methods this X-reducing substance will continue to show a few milligrams of reducing substance. In short, by their methods, including the recent modifications, it should not be possible to obtain a zero blood sugar.

Whether or not these modifications of Benedict's and Folin's give more nearly the true blood glucose content must await further work. The blood sugar value found during the insulin reaction to be reported strongly suggests that in this particular case all of the reducing substance was glucose.

The patient was an active boy of four years who had been a severe diabetic since the age of fifteen months. He had required insulin since the sudden onset of his diabetes and had been very difficult to control. Many combinations of diet, amount of insulin, and division of insulin dosage had demonstrated that if he were to be kept sugar free an insulin reaction at some time of the day or night was likely to occur. He had had many reactions of varying degrees of severity but had always responded quickly to the administration of orange juice. The reactions were apt to occur in the morning immediately upon waking even though the last previous dose of insulin had been at 6:00 P. M. of the day before. Such was the event during the reaction to be described. His last dose of insulin was 12 units at 6:30 P. M.

When the nurse entered his room at 7:30 A. M. to dress him she noticed that he was slightly stuporous and unable to sit up. She immediately gave him 100 cc. of orange juice, after which his strength and mental activities returned. He was dressed and given breakfast at 8:00 together with his morning dose of insulin (12 units). After one hour he appeared sleepy, got onto the bed, and remained there until 11:00, when I first saw him. I suspected an insulin reaction, so attempted to rouse him. Upon waking he was drowsy but irritable, recognized individuals, but spoke only a few words. Both eyes were drawn to the left and when voluntarily directed to the front were involuntarily pulled again to the left. There was no other evidence of muscle spasm. His skin was moist and cool with a pale and somewhat cyanotic color. Pulse was 135, but fair quality. A few minutes after being awakened the skin of his face became more pale. Finally he vomited a very large quantity of undigested material which represented his breakfast and the orange juice administered before. His color became better immediately and his stupor lessened. There seemed to be no reason for great apprehension and so another 100 cc. of orange juice was administered and then some of his dinner. After one-half hour his color again became very pale and somewhat cyanotic, followed soon by vomiting.

Having never before reacted to insulin in this manner, the question was entertained that a gastro-intestinal upset might account for a large part of the symptoms. To make sure, a blood specimen was obtained from the arm and examined quantitatively for sugar by the method of Folin and Wu[1]. The solutions and technique conformed in every detail to the directions given by these authors. There was not the slightest reduction of the alkaline copper tartrate solution during boiling or of the phosphate molybdate reagent when added. The blood sugar was zero. The determination was later repeated with the same result.

During the heating of the test it was seen that the blood sugar was very low and before the determination was finished the patient was given subcutaneously 0.3 cc. of adrenalin. Just after the adrenalin was given he exhibited a few twitchings of the muscles of his hands and arms. After fifteen minutes he suddenly brightened and took 100 cc. of 10 percent glucose solution. Within another ten minutes he asked to get up and when allowed to do so appeared as strong as usual. No further hypoglycaemic symptoms returned.

It appears that hypoglycaemia may be accompanied by such a depression of physiological activity that glucose by mouth will not be absorbed or passed on by the stomach.

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IF OUR MEDICAL WARRIORS WERE SLACKERS

WHAT if Gen. Gorgas had been a slacker and had thrown up the job of wiping out malaria in the Panama Canal zone? Would this, our great United States, have gained its present eminence in world power? And think of the lives that have been saved through his great sense of public duty!

If Jenner had been a slacker would he have discovered the preventive against smallpox that is saving uncounted numbers of lives through very simple and safe precaution?

If Pasteur had been a slacker, would he have found that germs are the cause of disease, and so, more or less, easily controlled? Would he, as a slacker, have found the cure for hydrophobia or rabies, that dread disease that has taken so many young lives?

There are any number of men who have risked their lives in the battle against disease and some have paid the supreme price for their sense of duty.—*Chicago's Health Bulletin.*

The Massachusetts Medical Society

THE CONTROL OF THE COMMUNICABLE DISEASES PREVALENT IN MASSACHUSETTS*

With a Study of the Mortality Due to Them During the Past Seventy-Five Years

BY EDWARD G. HUBER, M.D.

(Continued from page 634)

CONTENTS

12. EPIDEMIC CEREBROSPINAL MENINGITIS

In 1805, Vieusseux, in Geneva, studied an epidemic of epidemic cerebrospinal meningitis and decided it could be distinguished from the other disease with which it had previously been classified. That disease has since been further differentiated into typhus and typhoid. He came to his conclusion through autopsies, the only method of differentiation then possible. The next year, working independently, two Massachusetts doctors in Medfield, Danielson and Mann, came to the same conclusion, also by means of autopsies. Murchison in England disputed these claims, and the English followed his lead. Probably the disease had existed for centuries before this time, for various writers as far back as the sixteenth century described epidemics which at least suggest cerebrospinal meningitis. In 1810 there were so many cases in the villages of Massachusetts that a committee of the Medical Society studied and reported on the disease. During the War of 1812 the disease existed in the cantonments. It was known as spotted fever but probably included both bronchopneumonia and meningitis. During the Mexican War there were not many cases but it was widely disseminated afterwards. It was wide-spread during and after the Civil War. In 1866 a committee of the Medical Society studied 280 cases. In 1873 the disease prevailed under epidemic conditions but the year 1904-1907 saw the maximum distribution and mortality in the history of the disease.

The age distribution has varied greatly in different epidemics so there is no agreement as to what age group is most susceptible. In Massachusetts about half the deaths recorded under those diagnoses which probably include most of the cerebrospinal meningitis have been at ages under five, and three-fourths have been under twenty (table 11). So it is concluded that children and young adults are most susceptible. The sexes seem to be equally attacked.

The disease is most prevalent in the winter months and it thus corresponds in this respect to the other diseases supposed to be transmitted by naso-pharyngeal scretion. Cold is not a direct factor, for the disease occurs in the tropics, and if the meningococcus is exposed to a temperature of less than 22°C it promptly dies. But cold drives people more indoors, to closer contact with each other, and as catarrhal inflammations of the upper respiratory tract are more common in cold weather, the resultant sneezing, coughing, and spitting affords more opportunity to transmit infections, by the operation of chance alone. Besides, in cold weather there is more than a tendency to maintain too high a temperature indoors.

The etiological factor was proven in 1887, but had been suspected some years previously. The causative organism is non-pathogenic in the naso-pharynx where it is often found, but pathogenic to the meninges. Fortunately only a small proportion of those who receive infected material contract the disease, but the most vigorous and healthy individuals are often included in this minority. Lowered resistance seems to be a factor in susceptibility. War experiences very definitely point to the fact that the incidence of the disease was highest where recruits were suddenly plunged into a heavy schedule of work and training. Ordinarily the period of incubation is from two to ten days, oftenest seven. On the other hand it may be much longer, for individuals who have been carriers for some time occasionally develop the disease, and if the organism in the spinal fluid is the same type that he has "carried," his incubation period was relatively long. Vaughan thinks this depends on individual permeability of the walls of the naso-pharynx. Most persons have a natural immunity, even when they become contact carriers, but an attack does not seem to confer permanent immunity.

This disease has always been one largely of the barracks, and generally in time of war, for it is the recruit and not the seasoned sol-

*Published by the Committee on Public Health of the Massachusetts Medical Society.

dier who is the easy victim. Epidemics among crowded young individuals in civil life have not been unknown. Vaughan calls attention to the fact that up to 1865 all the reported epidemics were in rural regions. Since then, cities have been involved, although villages still contribute their share of epidemics and farmhouses their isolated cases. There are probably between 15,000 and 20,000 cases of the disease annually in the registration area of the United States. In the army during the World War there were 2988 cases, with 1179 deaths, a case fatality of 39.4%. Untreated with serum, this figure would probably have been about 70%. The average case fatality in the country is estimated to be about 50%.

Mortality statistics for cerebrospinal meningitis in Massachusetts cannot be given with sufficient accuracy to make them worth while. The disease has existed at least ever since Danielson and Mann did their historic work on it, but 1873 was the first year in which it appeared in the mortality records. In that year 747 deaths were ascribed to it, several times the subsequent annual number of deaths with this diagnosis. In 1897 there was a considerable epidemic in which 184 deaths were reported in Boston with many more in neighboring cities. This epidemic was the first extensive one after the discovery of the diplococcus in 1887, and as so little was known of the disease a commission consisting of Councilman, Mallory, and Wright was asked to study it and to make report. They made a careful clinical investigation of the cases in the principal hospitals and of others in which the diagnosis was certain. They doubted the accuracy of the statistics of the disease because of the confusion of medical terms by physicians, and because the disease was not common and was liable to be confused with other cerebral diseases or with those in which cerebral symptoms predominate. They found that from 1887 to 1895 26% of deaths from this disease were in infants, and concluded that this vitiated the accuracy of the figures for "the disease is extremely rare among infants as well as among those of advanced years." The reported deaths occurred mainly in large cities and towns, the whole number from towns smaller than 5000 being less than 5% of the total number. In the 1915 report of the Massachusetts Department of Public Health the diagnosis of many of the cases reported in the preceding years was stated to be doubtful owing to infrequent laboratory confirmation, and also because many of the histories showed that the meningitis occurred during pneumonia or tuberculosis. In the 1918 report still more skepticism was expressed, for a study of histories of cases showed that at least 50% of them were probably incorrectly diagnosed.

There has been more confusion in the terminology of this disease than in any other in Massachusetts. The following list includes those diagnoses which appear as causes of death under which actual cases of epidemic cerebrospinal meningitis might have been reported. Tuberculous meningitis is not included.

TABLE 11

1849-1854	Inflammation of brain
1855-1872	Cephalitis
1873-1900	Cephalitis
	Cerebrospinal meningitis
1901-1911	Meningitis simple
	Meningitis, epidemic, cerebrospinal
1912-1916	Meningitis simple
	Meningitis, epidemic, cerebrospinal, undefined
	Cerebrospinal fever
1917-1920	Cerebrospinal meningitis, undefined
	Meningitis simple
	Cerebrospinal fever
1921-	Non-epidemic cerebrospinal meningitis
	Meningitis simple
	Meningococcus meningitis

TABLE 11A

1849-1854	Inflammation of brain
1855-1900	Cephalitis
1901-	Meningitis simple

Total deaths	56,495	
Deaths under 5	36,019	64%
" 5-9	4,688	8%
" 10-14	1,998	4%
" 15-19	1,621	3%

TABLE 11B

1873-1900	Cerebrospinal meningitis
1901-1911	Meningitis, epidemic, cerebrospinal
1912-1916	Meningitis, epidemic, cerebrospinal, undefined
1917-1920	Cerebrospinal meningitis, undefined
1921-	Non-epidemic cerebrospinal meningitis

Total deaths	8,772	
Deaths under 5	4,500	51%
" 5-9	1,185	13%
" 10-14	659	7%
" 15-19	576	6%

TABLE 11C

1912-1920	Cerebrospinal fever
1921-	Meningococcus meningitis

Total deaths	363	
Deaths under 5	173	48%
" 5-9	40	11%
" 10-14	18	5%
" 15-19	32	9%

TABLE 11D

	Table 11A	Table 11B	Table 11C
Deaths under 5	64%	51%	48%
" 5-9	8%	13%	11%
" 10-14	4%	7%	5%
" 15-19	3%	6%	9%
	79%	77%	73%

In tables 11 A, B, C, and D are shown the numbers of reported deaths under the various

diagnoses which probably included most of the epidemic cerebrospinal meningitis. With such extreme diagnostic confusion it is not possible to make any but very general conclusions. The three arbitrarily chosen groups in A, B, C, and D have very similar age distributions but the actual amount of the specific disease included in them is very uncertain.

So far as is known the only source of the infecting organism available to produce the disease in another is the nasopharynx of a human being who is either ill with the disease or is a carrier. The infective material leaves such an individual either in droplets, or on saliva-moistened objects such as eating utensils. The virus must reach another person quickly if it is to find a new home, for it does not exist long outside the human body. The infection may be received in inspired air which contains infected droplets, or via food or eating utensils, or by contact with carriers or sick individuals. The last method is rare, however. It can be readily seen how crowding may increase the chances of receiving the infectious material in inspired air. Also when living conditions are crowded, messing facilities are likewise congested and eating utensils are insufficiently cleansed. The actual source of an infection is difficult to trace. Most of what has been said is based on circumstantial evidence and there probably is some unknown but important factor yet to be discovered other than crowding with its lack of ventilation, its overheating, and the increased opportunities for exchange of saliva. The literature does not mention any epidemics as occurring in schools.

The subject of meningococcus carriers is a very difficult one. It is generally agreed that the normal proportion of them is about two per cent of the population and that this figure may become much higher (10-20%) during an epidemic. In no other disease is the proportion of carriers to cases so high. But the very high percentages reported by some observers seem impossible. Zinsser calls attention to the possibility that such organisms as M. catarrhalis, pharyngis siccus, and M. flavus may have been mistaken for the meningococcus. However, the fact remains that so far as we know most cases are contracted from carriers and not from the actually sick. Infections among doctors and nurses who care for cases are comparatively rare. Vaughan and Palmer reported that of twelve cases at Camp Cody not one was associated with another. At Camp Beauregard all the 132 cases were independent of one another and there were no secondary cases in any organization. Carriers are more difficult to handle than in diphtheria where a virulence test is possible. They are numerous, and cases are few—five to one, according to Vaughan and Palmer—especially at the close of an epidemic. And since there are so many carriers and they exist in groups, it is the exception to trace a case to one. Carriers may be temporary or chronic. In the latter there may be a local focus of infection somewhere in the upper air passages. Carriers are obviously more important as sources of the infection than the sick, in whom the disease is recognized and whose activities are limited if by nothing else than the severity of the disease. Glover showed that the meningococcus carrier rate is a direct index of the degree of overcrowding and that when the space between beds is increased the carrier rate drops to normal. This rate shows a sharp rise just before an epidemic. He regards a rate of 20% as the danger line. Mink's data, from the Great Lakes Training Station are very conflicting and indicate no such rule.

The disease is an anomaly so far as its control is concerned. Its etiology is known and its mode of spread is understood but its control has been a failure. It has been shown to be useless to swab contacts by the thousand and then to isolate the carriers, for when the latter were segregated, new crops developed at once. The normally existing proportion of carriers is responsible for the sporadic cases, who are the occasionally found, very susceptible individuals. At times, instead of the cases remaining sporadic, an epidemic starts. Some cause, such as overcrowding, has increased the number of individuals who receive the meningococcus into their nasopharynges, or the organism or attendant conditions have changed so that virulence or ability to grow on a nasopharynx is enhanced. Then the epidemic develops.

The most important control measure is to seek and correct the underlying cause. If it is overcrowding, the remedy is to disperse the individuals. Time spent in searching for carriers is better spent in a study of the underlying conditions. Persons who have catarrhal symptoms and who sneeze, cough, or spit should be isolated at any time, epidemic or not, and general personal hygiene measures should be recommended in order to reduce catarrhal inflammations of the upper respiratory tract. This is not recommended with any idea that the meningococcus causes such symptoms but because an individual so infected has much greater proclivities for spreading his mucus. Overcrowding takes place in public conveyances during the entire year, but there is ventilation during warm weather. Similarly with public gatherings except theaters the majority of which are now satisfactorily ventilated during the entire year. Therefore, during an epidemic the public should be warned to avoid crowding.

Insufficiently washed dishes and eating utensils are probably an important factor, especially in camps, industrial organizations, restaurants, and boarding schools. These articles should be washed in clean water sufficiently hot

to kill pathogens. This is an excellent practice as a routine procedure.

The case itself should be isolated and concurrent disinfection practiced. As was said, secondary cases are very rare, but it is comparatively simple to isolate a sick individual and although as Hill says, isolation is seldom more than 80% perfect, and public sentiment would not allow a health officer to abandon it if he would, it causes but little inconvenience and may save an occasional infection. Contacts should of course be cultured, and those who are positive isolated and given special attention, such as spraying with dichloramine T in order to lessen the danger to their contacts. Release from isolation may be permitted when there have been two negative successive weekly swabs from the nasopharynx, or the epidemic is over. To attempt to isolate carriers on a large scale, according to type of organism, and according to whether the last swab was negative or not, becomes a very complicated process, impossible to carry out.

There is no need to quarantine other members of the household. Throat cultures should be taken in a search for carriers. The house should be placarded. The disease is, rightly, a notifiable one. But there are so many missed, unrecognized, and mild atypical cases that all the infections can never be known. The accurate diagnosis rests on a demonstration of the meningococcus in the spinal fluid, and a spinal puncture is not done as a routine outside of hospitals.

The public in general must be educated in personal cleanliness. There will be no control, Vaughan says, until "man becomes sufficiently well trained in personal hygiene that he is able to avoid receiving all consignments of bacterial flora from the upper air passages of his neighbors." When this is accomplished, "the last of the meningococci will die."

13. Anterior Poliomyelitis

There have been written during the past century and a half descriptions of a disease which we now conclude was anterior poliomyelitis. It was not always considered to be an entity, or was it suspected of being communicable until the early 80's of the past century. This suspicion was turned into conviction by Wickman's study of an epidemic of over 1000 cases in Sweden, in 1905. He recognized mild and abortive cases which did not progress to paralysis and was thus able to show that communicability was very probable, those links in the chain having been missing up to the time of his studies. No progress of any importance in the study of this disease from a public health standpoint has since been made.

Nothing is known of the nature of the virus except that it is filterable, that it exists in the excretions of the nasopharynx, several days before the clinical manifestations of the disease until about two weeks after the onset, that it occurs in the intestinal contents, and that the monkey is an excellent experimental animal. The disease is chiefly confined to the North Temperate Zone. Aycock and Eaton studied the monthly distribution of 54,238 cases reported in the United States Registration Area during the eleven years from 1912 to 1922. They showed a persistent and definite late summer prevalence, with a smaller secondary increase of cases in March or April. Over half of the cases occurred during the 1916 epidemic. If anterior poliomyelitis is transmitted chiefly by the nasopharyngeal secretions, the seasonal prevalence should be the same as the other diseases which are thought to be distributed that way,—namely pneumonia, influenza, and cerebrospinal meningitis. Aycock and Eaton interpret the lesser rise in the spring as being due to transmission in this way, and the greater one in late summer to be due to some unknown cause. Inhabitants of the smaller towns and the rural population seem to be attacked most frequently. Children become infected more frequently than adults. One attack confers immunity, according to experiments on the monkey. The period of incubation is about ten days. No method of immunization has yet been developed although the blood of a convalescent neutralizes the virus, before injection. Susceptibility is very variable. Frost says that of 2070 persons exposed to the disease by residence in the same house or family with a case of it, only 14 (0.6%) developed typical attacks of the disease; if, however, the atypical cases and those suspected of being poliomyelitis are included, the percent becomes 3.2. The disease is probably transmitted by contact, both direct and indirect, as well as in some unknown way. As the virus is not long-lived, indirect contact must be very recent. That the virus may enter the body through the nasopharynx and produce the disease has been proven by experiment. It has also been found in mesenteric lymph nodes, and it is therefore assumed that it may enter the body via food. In this connection, the mechanical transference of the virus by flies must be given some consideration.

Since carriers probably exist, and mild, atypical, abortive, and missed cases are so frequent, control is almost impossible at present. If the chief method of transmission is via the nasopharyngeal excretions, control depends considerably on prevention of the exchange of saliva. That carriers are important factors in the etiology of the sporadic cases may be assumed. The disease should be a notifiable one, and the sick individual should be isolated with concurrent disinfection of intestinal as well as nasopharyngeal excreta. Flies should be excluded from the sick room. Quarantine of contacts does not seem to be worth the incon-

venience it causes for there are so many unrecognized cases and carriers which are not isolated that the number of cases so transmitted must be relatively very insignificant.

14. Encephalitis Lethargica

The first definitely known epidemic of what is now called encephalitis lethargica occurred in Austria in the winter of 1916. During 1918 and 1919 the disease spread to the rest of Europe and to the United States. These manifestations of an apparently new disease caused a thorough search of the literature for evidences of previous visitations of any disease with similar symptoms. As a result of these studies it is thought that the disease known as "nona" which existed in northern Italy in 1890 may possibly have been encephalitis lethargica. There are also descriptions of outbreaks of disease in the late eighteenth and early nineteenth centuries which suggest the malady as it is now understood. Much attention has been accorded the disease in the last four or five years.

The well known work of Loewi and Strauss indicates that the cause of the disease is a filterable virus which is found in the central nervous system and in the excretions of the nasopharynx. Success in infecting animals has however been variable. The virus of herpes is found constantly in the mouths of some individuals and occasionally in others, and when this is injected into animals often produces an encephalitis. For a time a relation was believed to exist between these viruses, but that belief is now discredited. It is possible that the experimental encephalitis produced from nasopharyngeal secretions of cases of encephalitis lethargica may have been due to the virus of herpes. Nevertheless the disease is generally accepted as being a specific infectious one, although there are indications of the existence of some mysterious realtionship between influenza, anterior poliomyelitis, and encephalitis lethargica. These three diseases are all very vague as to their etiology and this ignorance must be cleared up before any great steps in control can be taken.

The disease is not common in childhood, or at least is seldom recognized at such ages. The sexes seem to be afflicted equally. If there is a definite seasonal incidence it is the winter months, although too few cases have been studied to be certain. The case fatality varies from 20 to 33% as reported by different observers, probably depending on how many mild or abortive cases are included in the basic figures. Kling, in 1920-1921, studied an epidemic in Sweden which occurred in small communities. He reports the morbidity to have been from 7 to 45% in the villages. In some families nearly all members were afflicted. He stresses the many abortive cases, which are important factors in transmission. He concluded that the period of incubation was ten days. On March 1, 1921, encephalitis lethargica was declared a disease dangerous to the public health, and reportable in Massachusetts. In 1921, 117 cases with 73 deaths were reported The next year 163 cases and 82 deaths were recorded.

Until more is known of this disease its control will be impossible. There is not even definite evidence that it is communicable. Those who consider it so, speak of the infectivity as low; in order to find evidence on which to base that conclusion a careful search of the literature is necessary, for comparatively few cases point definitely to contact with another case as the undoubted source of the infection. On the other hand, if the disease is communicable, it may be assumed that there are many mild, missed, and abortive cases, as well as carriers, who are able to transmit the virus. Inasmuch as there is no diagnostic criterion no one can say definitely when the disease exists. It should certainly be a reportable disease for purposes of study, but no other measures need be taken. If the patient is cared for in the ordinary cleanly manner no one runs an appreciable risk. There seems to be no necessity of keeping children who are contacts out of school.

15. The Venereal Diseases

Of the three venereal diseases, chancroid has very little public health significance and is not, therefore, being considered in this study. Syphilis and gonorrhea will be discussed together, since they do not differ materially from the standpoint of prevention, and since the details of treatment are not here being considered. Syphilis has always received much more attention than gonorrhea; the latter is still regarded by many as "no worse than a cold," and its history is not even as well known as is that of syphilis. Whether the latter originated in the New World or whether it had existed from time immemorial and had merely had a recrudesence in its virulence late in the fifteenth century is still a moot point. At any rate, when the method of transmission was recognized, during the fifteenth and sixteenth centuries, a great change took place in the attitude toward prostitution. There were two causes for this; the fear of disease, and the Reformation. Active measures to combat the venereal diseases were first taken by Denmark, in 1672, when treatment by quacks was forbidden. In 1750 French orders required that prostitutes committed to the workhouse must, if infected, be cured in hospital before beginning their sentences. About forty years later Denmark provided free hospital treatment for all cases and in 1859 penalties were placed upon those propagating the disease. Ten years later the study of these diseases was required of all students of medicine in that country, and in 1874 the com-

pulsory treatment of all infected individuals was ordered. In 1900 Credé's procedure also became compulsory. It is thus seen that Denmark has been the poineer in combatting the venereal diseases. Other countries have been slow to follow, for it was not until the exigencies of war required it that any extensive serious measures were taken in the United States. The second decade of this century saw the most progress in the study of syphilis, and the discoveries made during that period.—namely, the treponema, salvarsan, the application of the complement fixation test to syphilis, the value of calomel as a prophylactic, and proof that the parasyphilitic diseases were due to the treponema, form all the basis for control measures. In 1912 New York City's Board of Health took the lead in this country when it decided that the venereal diseases should be regarded in the same way as any other highly communicable and preventable disease. A few cities followed New York's lead, so that by the next year 7 of the 227 largest cities in the United States required the reporting of cases and four of them had free venereal disease clinics. This movement received such an impetus during the war—when the very wide prevalence of these diseases first received publicity—that by 1920, 82 of the 83 largest cities required reporting of cases and all but two had venereal disease clinics. This fight has been conducted by federal, state, county, and municipal authorities, as well as by numerous private agencies, and remarkable progress has been made. Owing to the fact that the venereal diseases can be combatted from so many different angles it is possible for a number of organizations to coöperate without friction, each having its own sphere of action which contributes its part to the whole. The campaign has not been without its difficulties which have been even greater than in the anti-tuberculosis fight, which offers certain analogies.

It is of course impossible to present any reliable figures as to mortality and morbidity for the venereal diseases. Syphilis and gonorrhea appear infrequently as causes of death both because of the stigma attaching to such a diagnosis and because it is only in recent years that the protean manifestations of syphilis have been recognized as such. If aneurism, general paralysis, and a large proportion of diseases of the brain, spinal cord, and heart were properly listed as syphilis, the latter would, as Osler said, rank with cancer, tuberculosis, and pneumonia as a cause of death. And those three diseases cause about a fourth of all deaths. As to morbidity, the best information concerning the prevalence of the venereal diseases is obtained from the examination of drafted men, and from the incidence in the Army. The infections found on admission to the Service were of course a result of conditions in civil life and may therefore be taken as an indication of their prevalence there. The data were so startling that efforts at control were at once initiated. Since the war there have been published more or less accurate morbidity statistics. According to the report of the Surgeon General, U. S. P. H. S. for 1921 the morbidity rate for the United States was 404.4. In the largest of the 83 cities the rate was 441.2; in the next largest group it was 422.1; in the smallest it was 782.7. It is very improbable that reporting is anywhere complete; probably the majority of reports received are from clinics. Higher morbidity rates probably mean nothing more than that the physicians are coöperating more in reporting cases. The literature contains numerous discussions as to whether the venereal diseases are increasing or decreasing, but all conclusions are based on insufficient data.

The problem of control is markedly different from that in the non-venereal communicable diseases. In the latter the actual cure of the infected is a matter of secondary importance from the public health point of view. But in the venereal diseases the cure is the primary consideration. Other communicable diseases are more or less self limited and cure or kill within a brief space of time. But the venereal diseases do not cure themselves and each case is a potentially active carrier. On the other hand the cure is available, so that ignorance and unwillingness to apply available knowledge are the chief reasons for the continued prevalence of these diseases. In a fight against a disease it is impossible to accomplish anything unless the majority of public sentiment is supporting the combat. The efficient health officer keeps a step ahead of the public. If he is too far ahead, or behind, he is useless. This is especially true in the venereal diseases for here we come in contact with the intimate life and morals of the people. It is impossible to obtain any change in morals by legislation, rather does legislation indicate the state of morals of a people. Morals may be influenced by education which is therefore one of the most powerful weapons in the anti-venereal fight. The public is not yet ready to attack the venereal diseases in the same manner that it is such diseases as small pox, malaria, tuberculosis, or diphtheria, for in the public mind the question of morals is closely bound to it. Milton said that the bulk of humanity "of love and love's delights take freely." This has been true for many centuries and therefore it is useless to direct the fight against the vehicle of transmission of the disease. The only sane procedure is to concentrate on prevention of infection. Purists insist that sexual intercourse should be only for the purpose of reproduction but such a state of affairs is impossible of fulfilment.

Gonorrhea and its complications present an

even more difficult problem than syphilis. Since notification of these diseases has been in practice there have been fewer cases of the former reported than of syphilis. This is contrary to the prevailing ideas as to the relative frequency of occurrence of these two diseases. In all probability this discrepancy is due to the fact that gonorrhea is not considered as important as syphilis and therefore is not so generally reported. Gonorrhea is not so fatal as syphilis but its cure is more difficult and carriers are both relatively and absolutely greater in number. The public must realize that as Osler said it is "a great communicable disease, many of whose victims are innocent."

The source of infection, the mode of transmission, the incubation period, and the period of communicability for the venereal diseases are perfectly well known, beyond any doubt; therefore the anti-venereal disease fight is not handicapped by any uncertainty as to these basic points. The necessary steps are clear but the possibility of making full application of available knowledge seems remote. Looking into the future, the education of the public is even more important in the venereal diseases than in the other communicable diseases. The first essential in control measures is similar to that in the other communicable diseases, namely, prompt recognition followed by reporting. As an aid to early diagnosis the state laboratory is invaluable. It is also a check on reporting. Notification is of real practical value which is not true of such diseases as varicella and measles. The case should be reported by office number when treatment is instituted, and not by name and address until the patient fails to continue treatment as directed. If the patient follows the advice given him as well as could be expected, there is no need of the health authorities knowing his identity, but once he stops treatment he becomes a neglected carrier, liable to remain so for an indefinite period, and a direct concern of the health department. Reports giving name and address should be sealed. A regulation requiring the laboratory confirmation of each clinical diagnosis seems wise not only in justice to the patient but as a check on reporting.

Isolation in the venereal diseases is not necessarily the same as in the other communicable diseases. It may vary from simple instructions from the physician to his intelligent patient, to forcible detention of the ignorant or wilful. Legislation is necessary in order to be able to isolate those who refuse treatment and presumably spread their infection. Such a law does not need frequent invocation, for tact and persuasion often accomplish all that is needed. The fact that such a law exists has a beneficent influence, however. Besides forcible detention, adequate treatment facilities must be provided. If the infected person refrains from sexual intercourse and the preparation or preservation of food, the general public will be protected. He must also avoid infecting the other members of his family, using separate dishes, towels, etc. In other diseases carriers are more or less numerous but we are powerless to cure them. In the venereal diseases every case is a carrier, and in syphilis at least, the cure is certain. But it must be made easy for the infected person to get proper treatment. The majority of physicians are neither willing nor prepared to treat these diseases, but they should be able to refer such cases to reliable practitioners where they can be efficiently treated and advised. Every patient able to pay should be under the care of a competent physician; the latter is the bulwark of the battle against the venereal diseases as well as against all the other communicable diseases. Early and skilful treatment diminishes the sources of infections. In order to assist physicians in their intelligent and effective handling of these diseases four measures must be undertaken: the suppression of quacks, the removal of patent medicines from the market, prohibition of advertising of such nostrums, and cessation of druggists' prescriptions and treatments. Legislation is needed for all four, as a background. Prosecution will be needed for the quacks, but a campaign of education for the druggists as conducted by Kelley in Massachusetts can be successful without a specific law. Druggists were personally seen and when the matter was explained to them were practically all willing, and did co-operate. Applicants for treatment at a drug store should be informed by the druggist where free clinics or qualified physicians may be found. It is of no avail to abolish quacks and patent medicines if there are insufficient facilities for correct treatment. There must be provided detention homes, and venereal wards in general hospitals, for the quarantine of the refractory as well as dispensaries and clinics for those willing to take treatment but not able to pay a physician. Care must be taken that none who are able to pay are given free treatment. The co-operation between physician and clinic must be complete, and this will be impossible if the clinic treats those who should employ a physician. These clinics may be maintained by the health department or other municipal department, by federal, state, or county funds, or by private agencies; they must be so located that patients may visit them with a minimum of observation by the curious.

Care should be exercised in reporting patients as cured. It would be safer never to discharge cases as cured but to release them from treatment and then only after a negative spinal fluid Wassermann test. Following this, they should return for several semi-annual blood Wassermann tests. Cures will not only diminish disease incidence but will lower the

mortality rates for the diseases of infancy and of middle life. In rural regions treatment facilities are badly needed. On admission to the Army, men from Alabama, Mississippi, and Florida were found to be infected to the extent of ten to fifteen percent. The only remedy is for the rural practitioner to prepare himself to take care of such cases to prevent their neglect or ignorant handling. Patients must be persuaded that a long time is needed for a cure, for the tendency is to be delinquent once the open lesions have disappeared. Here, follow-up work by letters, or personal visits, or both, may be very effective if tact is used. In the course of this routine infected contacts will often be located just as in anti-tuberculosis work. In selected families, instruction may be given as to how to avoid infection.

State penal institutions and hospitals should be prepared to examine all inmates and to treat where necessary. Complete permanent records should be kept for future use. Social service work among the members of the families of these inmates can be fitted in with the follow-up work from the clinics. Orphanages should be searched for those congenitally infected.

Co-operation with courts is also productive of excellent results. Here the health department is largely dependent on those agencies which are concerned particularly with the moral and social aspects of the question. The arrest of sex offenders should be immediately followed, before arraignment, by a physical examination. Discovery of a venereal disease should result in compulsory treatment whether the person is convicted or not, for such cases are most dangerous to the public health. Such detention and treatment should extend through the period of infectiousness regardless of the length of the sentence. There is an excellent field for the voluntary organizations in following up such sex offenders after their release.

The pre-natal clinics should, as a routine measure, examine for, and treat thoroughly when found, venereal diseases in prospective mothers. Such treatment is doubly efficacious.

It is often possible to ascertain the source of an infection by tactful questioning. The names of such individuals, their address, or any available scrap of information should be reported to some voluntary organization prepared to make tactful investigations, remembering that such names may have been given falsely and maliciously. Infected individuals thus found can generally be placed in charge of private physicians unless they are incorrigible, when the clinic or detention ward must be used.

Health departments should be empowered to examine food handlers and others such as barbers and beauty parlor operators and to exclude the infected until the period of communicability has passed. Such individuals will be faithful in treatment for they will desire to return to their employment. Routine examinations of this nature cannot wholly prevent the employment of infected persons, for the examinations cannot be sufficiently frequent. An examination at the beginning of employment and further ones at six month intervals is probably all that could be expected.

Certain legislation is necessary even if enforcement is seldom actually used. A model code has been suggested by the U. S. P. H. S. and with modifications required locally should be on all statute books. Rhode Island has a law prohibiting knowingly exposing another person to a communicable disease. The maximum punishment is a fine of $100 and three months imprisonment.

The eradication of prostitution, "the oldest profession," is a dream, but this does not mean that no attempts should be made to curb it. Each success in limiting this institution serves to diminish the venereal diseases.

Individual prophylaxis is opposed by many on the ground that it encourages promiscuity and that this increases the disease rate or at least the number of cases. Such arguments are based on the idea that the fear of disease is a great deterrent to exposure. As a matter of fact this fear has but a slight effect in preventing intercourse. In spite of all the preaching of reformers, moral persuasion, athletics, sex education, and advice to burn "fires on other altars than that of Venus" continence will not be the vogue among the adult population. If individuals expose themselves it should be possible for them to be able to purchase individual disinfection packets of calomel ointment which have received the approval of the state health department. It should be as easy to obtain such a packet as to procure sheaths. The availability of this packet is no more a temptation to get infected than is passive immunization in diphtheria an encouragement to expose oneself to diphtheria.

The measure which will ultimately have the greatest effect on the diminution of the venereal diseases is the education of the public. Sex education of the young is probably most important and requires the utmost care. The various methods of spreading information have been the subjects for many exhaustive articles in the literature, and will not be considered in detail here. Health departments should also keep physicians fully informed as to the new reliable procedures in treatment methods by distributing reprints as well as by sending representatives to the society meetings. Lectures with lantern slides and films, and pamphlets also have their field of usefulness in a general educational campaign. The health department may also spread information through employers, through schools, through clubs, through the ministry, and by encouraging certificates of family physicians (of the bride) before mar-

riage. In New York a statement has been incorporated in the Domestic Relations Act warning applicants for a marriage license of the necessity for making certain that they are not infected with a venereal disease. Education of the public will not merely help individuals to help themselves, but it will cause many of them to array themselves among the members of the community who are active in influencing public opinion, and in obtaining needed legislation, and in negating the influence of charlatans.

(To be continued)

TABLES

MEDICAL PROGRESS

PROGRESS IN UROLOGY

BY HERBERT H. HOWARD, M.D.

THE Probable Stimulation in the Rate of Growth of a Cancer of the Bladder by Deep X-ray Therapy[1]. Martin states that he has never seen a cure of cancer of the bladder by deep X-ray treatment, in spite of the fact that several apparent cures in France were reported, and he discusses in this paper a case in which it appeared that the growth of the cancer was stimulated by X-ray treatment.

At the time of the first examination a cancer about the size of a dime was found in the fundus of the bladder, the patient having had intermittent hematuria and dysuria for about four months. Operation was advised but refused, and the patient was treated by an experienced radiologist. At first there was some apparent improvement, then the bladder symptoms became much more pronounced and a cystotomy became necessary. Death occurred at the end of a year. At autopsy an extremely malignant type of carcinoma was found.

He considers that the growth, at the time of the first examination, was very favorable for operation and that it is unusual for this type of tumor to cause death so quickly.

H. C. Bumpus, Jr.[2], at the Mayo Clinic advises frequent re-examination of patients treated for tumors of the bladder, because of high incidence of recurrence.

He does not believe that radium alone is successful in malignancy of the bladder, especially when other methods of treatment can be used.

Radium is most useful in association with fulguration or operation, but radium treatment followed by operation gives poor results. If radium has been applied to malignant growths, the treatment should not be changed later to surgery.

Tumors of a low degree of malignancy that are too extensive to fulgurate or excise, are best treated with cautery.

Cauterization is not applicable to tumors of higher degrees of malignancy; these should be treated by excision or resection. The most important factor determining the final results of any type of treatment is the degree of malignancy of the tumor.

A. L. Dean, Jr.[3], in a paper on the Treatment of Teratoid Tumors of the Testis with Radium and the X-ray gives the following conclusions:

1. Teratoma testis is a disease associated with a high mortality. In the majority of cases inoperable metastases occur relatively early.
2. Because the differential diagnosis is difficult, all intrascrotal swellings must be carefully examined with the possibility of malignancy in mind. Serological and roentgenological data are frequently of value.
3. A favorable prognosis depends upon prompt as well as efficient treatment.
4. Simple orchidectomy alone usually does not give permanent relief.
5. Pre-operative irradiation of the primary tumor is most essential. Microscopic evidence demonstrates that the growth properties of the malignant cell may be destroyed. In cases given such preliminary treatment the mortality was 25 per cent lower than in those not given such treatment.
6. With adequate X-ray facilities at hand, the radical dissection of the lumbar and pelvic lymphatics seems to be an unnecessary surgical procedure.
7. The high-voltage X-ray has been found to possess certain advantages over the radium pack, and no disadvantages have been noted in its use.
8. Of seven patients with operable tumors who were treated, five (71 per cent) are living and apparently free from the disease. Of forty-nine patients who were first seen with inoperable metastases, ten (20 per cent) are now free from signs of the disease. External irradiation alone was used in this group of cases.
9. While the number of so-called five-year cures so far obtained has not been large, the fact that practically every patient was benefited to an appreciable degree suggests that when the technique and management become more nearly perfect the results will demonstrate more clearly the efficacy of physical agents in the treatment of teratoid tumors of the testis.

B. S. Barringer[4] classifies carcinoma of the bladder into two groups: cases of papillary carcinoma and cases of infiltrating carcinoma. These groups include all cases in which the tumor was removed intravesically up to June, 1919, when suprapubic implantation was begun, and cases in which it was removed intravesically or suprapubically since June, 1919.

Of twenty-three cases of papillary carcinoma of the bladder, the carcinoma was removed in fifteen. In six the result has not yet been determined. One patient is dead and another is dying.

Of sixty-one patients who had an infiltrating carcinoma, eighteen are well and thirty are dead or dying. The result is undetermined in thirteen cases.

A large majority of both papillary and infiltrating carcinomata have involved the trigone and the neck of the bladder. Of fifteen cases of papillary carcinoma the tumor was removed intravesically in twelve and suprapupically in three. Of eighteen cases of infiltrating carcinoma the tumor was removed intravesically in six and suprapubically in twelve.

The operative mortality of the suprapubic implantation of radium in infiltrating carcinoma is less than 2 per cent.

H. Oliverona[5] discusses the So-Called Uraemia Following Prostatectomy. He believes that in deaths coming within forty-eight hours following the operation the diagnosis of uraemia is erroneous because a true uraemia does not cause death until after several days. The diagnosis of uraemia in cases of prostatectomy is usually founded on the presence of oliguria or anuria, but the suppression of urinary secretion may be due to circulatory disturbances.

He reports two cases of anuria following prostatectomy. In both the clinical course is very similar. Immediately following the operation the patient's condition remained good for several hours, but on the second day there was a rapid pulse with cyanosis, coldness of the skin and oliguria. Finally there was complete suppression of urinary secretion. The first patient died at the end of thirty-six hours. The second patient was transfused and following the transfusion 300 c.cm. of urine was secreted in four hours. The amount of bleeding in both cases was only moderate.

He believes that the cause of death in such cases is shock by tissue injury, loss of blood, and possible infection and that the treatment in all of these so-called uraemia cases should be transfusion.

N. F. Ockerblad[6] discusses the Practical Application of the Creatinin Renal Function Test, and believes that it is a more definite test than the phenolsulphonephthalein test and has adopted the following procedure:

The patient is prepared for cystoscopy in the usual way after having been given from 1,500 to 2,000 c.cm. of fluid about half an hour previous. Urethra catheters are then introduced and when the flow is established the time is noted and collection is made for exactly ten minutes. The tubes containing the urine are then marked "fasting ten minutes." After the removal of the tubes 0.25 gm. of creatinin in 5 c.cm. of buffer solution, mixed with 1 c.cm. of phenolsulphonephthalein, is injected intravenously. At the completion of the injection, the exact time is noted, the tubes are replaced under the catheters and collection of urine is made for another ten minutes and as many ten minute collections as desired is made.

His experience shows that one fasting ten-minute collection and one ten-minute collection period are sufficient to give information of great value.

The advantages of this method are summarized as follows:

1. The complete elimination of the substance without chemical change by the kidneys.
2. The early appearance of creatinin in the urine following its intravenous administration.
3. The rapid excretion of creatinin, which necessitates observation for only a short time.
4. The brilliancy of color which is imparted to the urine when it is prepared for estimation by the method of Folin. Blood does not interfere.
5. The facility with which creatinin lends itself to colorimetric methods, making accurate estimation possible.
6. The simplicity of the technique for quantitative estimation.
7. The non-toxicity of creatinin.
8. The non-irritating nature of creatinin locally.
9. The extreme smallness of the dose required and the assurance this gives that no extra strain will be placed upon the kidneys during the test.

Creatinin is a normal constituent of the blood and an end-product of catabolism. So far as is known, it is neither destroyed nor stored up by any organ. The creatinin test is intended to supplement the phenolsulphonephthalein test.

F. G. Bell[7], in the *British Journal of Surgery*, discusses the teratoid group of tumors of the testicle and states that the majority of testicular tumors belong to this group and that the term "teratoma" should be used only in the high complexed types displaying structures which can reasonably be interpreted as a successful attempt on the part of the tumor to produce adult or fully formed structure or organs such as the alimentary tract, the bones, the kidneys, the breasts and spinal cords. He applies the term "teratoid" to the common, less complex

types in which the activities of the tumor are abortive and produce only a mixture of tissues.

J. K. Hollaway and W. H. VonLackum[8] believe that the prostate gland is infected in a large percentage of males with no previous history of gonorrhea. The infection is frequently responsible for numerous local and general symptoms and also for conditions classified as of focal origin. Prostatitis is latent and can be diagnosed only after repeated examination of the prostatic fluid. Systematic treatment of prostatitis and vesiculitis, consisting of the usual course of treatment, vigorously and systematically carried out, is in most instances followed by definite improvement.

L. P. Player[9] discusses the prostate and its influence on low back pain and in his summary states that pain in the back, until recently, was attributed in most cases to static conditions and nerve involvement in certain cases of arthritis.

Rosenow and his followers have shown that bacteria from foci of infection have a selective action and that the different types produce arthritis, synovitis, myalgia, and myositis. That eradication of focal infection in the prostate and seminal vesicles as well as in the tonsils, teeth, etc., later cure these troubles.

In prostatitis there are organisms analogous to those found by Rosenow in other foci.

He emphasizes the fact that it is necessary to massage the prostate and strip the vesicles from one to four times on consecutive days.

S. G. Jones[10] reports a new method of searching for tubercle bacilli in the urine. It is a modification of the dilution method. The technique is as follows:

The urine is obtained by catheter from the badder or kidney. A tube of urine is centrifugalized at a low speed for two or three minutes to remove the bulk of the pus and detritus. The supernatant cloudy fluid, containing a few pus cells and bacilli, is poured off, one-half is discarded, and the remaining half is poured into a second centrifuge tube. To the half-filled tube, one-quarter volume of 95 per cent alcohol and one-quarter volume of distilled water are added. This mixture is then centrifugalized at the highest speed for forty-five minutes until is clear, the supernatant fluid is discarded, and a smear is made from the sediment obtained with a flamed wire loop. The smear is allowed to dry and is then fixed by being passed rapidly two or three times through a Bunsen flame.

The centrifuge must be an electrically driven high-speed machine. When carrying a load of four tubes, it should make from 2,000 to 2,100 revolutions per minute, this producing a force 1,077 times that of gravity.

The Ziehl-Neelson stain is employed.

M. Negro[11], Pyelography in Renal Tuberculosis. Surgeons of note have expressed conflicting views regarding the value of pyelography in the diagnosis of renal tuberculosis. The author's conclusions on the subject are based on fourteen cases.

In the early cases in which the lesions consist of discrete tubercles in the parenchyma or pelvis, a pyelogram is negative in all respects.

It is essentially negative also in the so-called closed cases, although the parenchyma of the kidney may contain large abscesses and the pelvis and ureter may be considerably dilated. In these cases the papillae retain their normal outline and there is nothing pathognomonic of tuberculosis in the changes in the renal pelvis and the ureter.

The changes revealed by the pyelogram in tuberculous pyelitis are of only relative significance. In hydronephrosis the shadow shows distinct regular borders, but in some instances a faulty mixture of the opaque solution and the urine may be confusing. There is little to distinguish the picture from that of pyonephrosis.

In cases with ulcer and caseation, the ureter is straightened and dilated and the pelvis shows dilatation and the outline of a segment of cauliflower, but an accurate estimate of the extent of the disease is often impossible. Cavities filled with thick pus are not seen. Simple hydronephrosis, pyonephrosis, and stone may produce a similar picture. Therefore pyelograhy in these conditions is of only relative value.

In view of the difficulties and limitations of the method and the relative precision of other means of exploration, the author is inclined to agree with Marion that in renal tuberculosis pyelography is of little aid.

Eisendrath, Katz, and Glasser[12], in a series of ninety-one cases studied by pyelograms to determine the frequency and cause of regurgitation of the bladder contents into the ureters and renal pelvis, find the condition was present in five cases. There were three cases of non-obstructive bladder and prostatic lesions and two cases of renal ureteral lesions before operation. In twelve cases of bladder neck obstruction not operated upon, it was absent, although this is the type of case in which it could reasonably be expected to occur.

Their conclusions are as follows: Bladder reflux may be congenital or acquired. That it is not a permanent affection was shown by a case of atony of the bladder due to spinal syphilis in which it ceased after proper and general treatment.

They believe that bladder reflux plays an important part in carrying the infection from the lower to the upper urinary tract. The condition may lead to an erroneous diagnosis of

bilateral renal tuberculosis because it is a frequent complication of that disease. Acute and chronic cystitis prevent proper closure of the orifice on either one side or both and are important factors concerning bladder reflux.

Considerable difference of opinion still exists as to why the mechanism at the ureterovesical juncture, which normally opposes the reflux of the bladder contents, should fail to function. There are three theories advanced to explain this: First, a hypertonic vesical musculature forces the uretheral orifices open. Second, a congenital insufficiency. Third, the proper closure of the orifices is prevented in the wall of the lowermost portion of the ureter from lack of innervation or disease. They believe that all three may be determining factors in certain number of cases, or that each one alone may be sufficient cause for the reflux.

Gayet and Cibert[13]. These authors call attention to the work of Legueu, and also reports of diverticula of the bladder in recent literature and a review of twenty-two cases of their own.

From their study it would seem that the relative frequency of congenital and acquired diverticula has been reversed; the acquired type, they believe, being the more common. In seventeen of the authors' twenty-two cases the cause is apparently obstruction. In the others, the diverticulum was probably congenital. In one of the latter, in which there were two diverticula, the kidney, ureter, seminal vesicle, and vas deferens were missing on the side of the diverticula.

The congenital type occurs most commonly in an area just above and to the outside of the ureter where, in embryonic life, the mesonephric duct enters the cloaca. The condition is difficult to treat.

Four of the authors' patients were treated by lavage of the cavity without operation, three died. In three cases of diverticulum due to stricture, dilatation and lavage were followed by recovery. Of the three patients treated by cystostomy, two survived. Six treated by prostatectomy, four recovered. One case was treated by Young's method and three of four resections by the standard operations were successful.

The uninfected congenital type is ideal for radical treatment. Those, however, that are complicated by pathological changes are difficult. They believe, however, that radical treatment should be used whenever possible.

Beer[14] reports an interesting case of a man who had previously been operated upon by another surgeon for prostatic adenoma and bladder stone. Following the operation, however, symptoms recurred. X-ray and cystogram, taken by Beer, showed a dumb-bell calculus, one portion of which was intravesical and the other in a diverticulum. Evidently at the first operation the prostate and the intravesical portion of the calculus had been removed, but the latter re-formed. His treatment consisted of treating the neck of the diverticulum and removing the stone, followed by excision and obliteration of the diverticulum.

Negro and Blanc[15] discuss thirteen cases of diverticulum of the bladder together with the X-ray findings.

Most bladder diverticula, according to these authors, are silent and are discovered only at cystoscopy or at operation for a supposedly different urinary condition.

In three of their cases, the condition was noted in patients under forty years of age. Only two of the series were women.

In seven cases the diverticulum was paraurethral. In three, it was in the superior lateral wall. Two at the base of the bladder. One on the superior wall. In either the diverticula were single. The largest was the size of a large orange. Some communicated with the bladder freely and were sessile, while others communicated with it by only a narrow channel. In one case there was hematuria. In one the condition was associated with pyuria and a subumbilical tumor. Two of their cases contained stones.

The operative treatment indicated depends on the situation of the diverticulum.

In a diverticulum at the top of the bladder, they advise resection down to the bladder wall and drainage of the bladder.

For a diverticulum at the base of the bladder, the section through the neck of the diverticulum down to the neck of the bladder and the establishment of the largest possible opening between the bladder and the diverticulum.

For a diverticulum on the lateral wall, the dissection of the lateral wall of the bladder up to the edge of the diverticulum and resection at this point.

Day and Martin[16] state that, as a rule, two etiological factors are operative in diverticula of the bladder: (1) embryological defects in the bladder (weakened spots), and (2) obstruction (usually at the bladder neck). The modifying factors include situation (especially in relation to drainage), the size of the orifice, the size of the sac itself, the number of the sacs, the presence and type of infection in various parts of the urinary tract, focal infections, the nature and extent of the obstruction, secondary inflammatory changes in and around the diverticulum, calculus formation, and carcinoma.

In from 75 to 80 per cent of the cases the orifice is located near the ureteral opening and as obstruction of the bladder neck progresses, the gradual increase in the size of the diverticular sac may be observed in cystograms. When the orifice is very small the patient suffers in-

tensely from infection and inflammation. In these classical types the walls are essentially fibrotic. In every case with a diverticulum of considerable size opening near the ureteral orifice and well-developed prostatism the sac is rigidly adherent and may extend to the subpubic ligament subtrigonally. There is usually poor drainage with stasis, infection, and occasionaly calculus formation, especially in the presence of urea-splitting organisms, such as streptococci, staphylococci, or bacillus proteus, which render the urine ammoniacal. Haematuria is probably due to an abrasion or tear around the fibrotic orifice incident to spasm of the musculature peripheral to the neck. In advanced cases drainage can occur only by gravity and intra-abdominal and peridiverticular pressure.

The relatively rare forms that require excision are: (1) the hour-glass bladder, and (2) the large singular diverticula at or near the dome or on the lateral aspect high up near the vertex, with the wall composed grossly of all of the coats; this muscular wall is merged into a semifibrous ring surrounding the orifice. Cystography is apt to mislead as to the degree of retension that may occur even with dependent drainage.

In cases of diverticula of the anterosuperior dome with thick muscular coats the orifice is a most important factor. The position of the opening tends to favor drainage, but when an inflammatory exudate occurs, the bladder irritability and intolerance of distention begin, and the swollen and contracted muscle at or around the neck shuts off communication with the bladder. This seems to be a very late occurrence, but progresses rapidly when it once reaches a certain stage. On the other hand, the posterior diverticula are not capable of true contraction, but simply collapse; intra-abdominal pressure may partially empty them.

A rarer but important type is the small fibrous diverticulum which is usually situated just above and very close to the ureteral orifice. Two cases of this type showed hydronephrosis which could not be accounted for in any other way.

The following conclusions are drawn:

1. Nearly every diverticulum with marked clinical symptoms is associated with prostatism, either hypertrophy or contracture of the neck of the baladder. Rarely, the symptoms may be caused by ureteral obstruction.

2. Surgical relief of the obstruction is necessary in almost every case.

3. Excision of the diverticulum is indicated in cases of well-developed sacs of moderate or large size, if drainage is poor.

4. Advanced degenerative changes in the cardio-vascular system and kidneys may render excision of the sac itself inadvisable unless the cystitis is intolerable.

Hinman and Hepler[17] discuss Experimental Hydronephrosis: The Effect of Changes in Blood Pressure and Blood Flow on Its Rate of Development. Splanchnotomy: Increased Intrarenal Blood Pressure and Flow; Diuresis.

In the experiments reported the left ureter of rabbits was divided between ligatures about 4 cm. below the renal pelvis and in one group of animals the splanchnics were radically divided at the same time. The animals were then given a regular allowance of food and water and were killed at varying intervals up to ninety days after the operation. To determine the gross changes in the kidney, stereoscopic roentgenograms were made after the intra-arterial injection of barium sulphate, and the capacity of the renal pelvis was measured. The kidneys were then sectioned and studied further. The findings are summarized as follows:

1. Complete obstruction of a ureter in rabbits was followed by the progressive development of hydronephrosis. The degree of hydronephrotic atrophy was proportional to the duration of the obstruction.

2. Unilateral splanchnotomy caused a diuresis on the side operated upon as the result of intrarenal vasodilatation with an increase in the flow of blood through the kidney. This diuresis persisted for long periods.

3. The performance of unilateral splanchnotomy after complete ligation of the ureter had no influence upon the rate of development of the hydronephrosis.

Hinman and Hepler[18] discuss Experimental Hydronephrosis: The Effect of Changes in Blood Pressure and in Blood Flow on Its Rate of Development.

This article gives the results noted in a second series of experiments by the authors. In a previous publication they reported the results of unilateral splanchnotomy which produces an increase in the blood flow and intrarenal blood pressure. The rate of urinary secretion resulting from this procedure with complete ureteral obstruction influences the rate of development of hydronephrosis very little, if at all. In studies reported in this article the authors employed partial compression of the renal artery, which diminishes the blood flow and intrarenal blood pressure and thereby reduces the amount of urine secreted. These and the previous studies of the effect of splanchnotomy indicate that undue importance has been given to urinary back-pressure as a factor determining the degree of hydronephrosis developing after ureteral obstruction. Other experimenters have shown that partial compression of the renal artery produces a diminution in urinary secre-

tion due to cellular anaemia and a decrease in the blood flow and blood pressure.

The authors' experiments were performed upon dogs. Through a loin incision the kidney was exposed, the ureter was divided between ligatures, and the renal artery was isolated and then compressed by a special technique. The dogs were killed seven, fourteen, twenty-one, twenty-eight, and fifty-six days after the operation. In each instance the kidney was found enlarged, and in the later periods the enlargement was marked. The venous collaterals were greatly dilated. The rate of hydronephrotic atrophy and dilatation was constant and progressive throughout the series.

On histological examination of the kidney few atrophic glomeruli were found, fibrosis and connective tissue substitution predominating.

The conclusions drawn from these experiments are that back-pressure and pressure atrophy are of less importance than a nutritional factor. The more rapid parenchymal degeneration that results from arterial compression weakens the resistance and accelerates dilatation in spite of diminution and back-pressure.

Hager and Magath[19] discuss The Etiology of Incrusted Cystitis with Alkaline Urine.

From their study and experiments, Hager and Magath conclude that the inorganic salts which are deposited in the mucous membrane in cases of incrusted cystitis with alkaline urine are due to a secondary invader, a gram-negative bacillus, which in its growth liberates an enzyme, a urease capable of converting urea into ammonia and thereby rendering the urine alkaline. The resulting alkalinity precipitates the calcium, magnesium, and ammonium salts. The authors believe that the process may be superimposed on any type of bladder lesion. The initial lesion may be produced by some process which has not been investigated, but probably is often formed from a focus of infection, as Rosenow, Meisser, and Bumpus have demonstrated. Perez' bacillus, which is credited with producing crusts and a foul odor in the nose, also has the ability to convert urea into ammonia.

Since encrusted cystitis is far more common in women than in men, and since the organisms of the salmonella group are common inhabitants of the intestinal tract, Hager and Magath believe it not unlikely that the source of salmonella ammoniae is the gastro-intestinal tract and that the disease is caused by the implantation of this organism on a bladder lesion. This conclusion appears justified also by the fact that, in the cases of women, the history often dates from childbirth or catheterization. The authors were successful in isolating the organism once from a culture of a stool.

Braasch and Carman[20] discuss The Pyelographic and Roentgenological Diagnosis of Renal Tumors.

In cases of renal tumor simple roentgenography may reveal; (1) symmetrical enlargement of the renal shadow, (2) local projection of its contour, (3) areas of moderately increased density within a renal shadow of normal size and form, (4) partial or complete concealment of the renal shadow by the shadow of the tumor, or (5) localized densities due to calcified areas in the tumor, or associated calculi.

The presence of fimbriated streaks of calcification, noted in hypernephroma, is the only one of the manifestations mentioned which is considered pathognomic of renal tumor. The first clue to a renal tumor may be furnished by roentgenograms of the chest showing metastasis. Pyelography easily takes first rank among methods of demonstrating changes in the pelvic shadow resulting from neoplasms and cysts. These changes include: (1) elongation of one or more calyces or of the true pelvis; (2) shortening of the calyces; (3) encroachment on the pelvic lumen causing flattening of the pelvic contour, narrowing of individual calyces, obliteration of one or more calyces or obliteration of the true pelvis; (4) broadening of the calyces; (5) pyelectasis; (6) displacement of the pelvis; and (7) deformity of the uretero-pelvic juncture.

In the differential diagnosis thorough familiarity with the normal pyelogram and its variations is essential. It is possible to distinguish the different types of tumor. In cases of hypernephroma, which is possibly more common in the upper calyces, the calyces are deformed rather than the true pelvis and the primary roentgenogram may reveal calcified miliary streaks. The clinical triad of pain, palpable tumor, and haematuria is classic. In cases of carcinoma the pelvis is likely to be irregularly enlarged by sloughing of tissues, and the clinical triad is not marked. A short history and marked deformity of the pelvis suggest adenocarcinoma. In cases of polycystic kidney, abnormalities of the pyelogram are less constant and elongation of the calyces is infrequent and likely to be associated with widening of the calyx. Solitary cysts may cause shortening of the adjacent calyces and flattening of the nearest portion of the true pelvis. The weight of a cyst at either pole may change the axis of the kidney.

Benign neoplasms are rare and have no marks of distinction from other renal tumors.

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PRACTICE OF ANY BRANCH OF MEDICINE BY OTHER THAN LICENSED PHYSICIANS OPPOSED BY *MEDICAL WEEK*

THE *New York Medical Week*, official organ of the Medical Society of the County of New York, in discussing a legislative act proposed some months ago relative to the proper qualifications of anesthetists, declares editorially that the practice of any branch of medicine by other than licensed physicians is not only detrimental to the advancement of scientific study but to the best interests of the patient as well. The bill in question sought to limit the administration of anesthesia to licensed practitioners.

"It was surprising," says the writer, "to note how many physicians and hospitals were opposed to the project."

"At this time it is almost platitudinous," he continues, "to assert that modern surgery owes the tremendous strides that it has made largely to the development of anesthesia. Aside from the procedures he makes possible, the anesthetist today is the guardian of the general physical condition of the patient during surgery. It is true that in many simple, uncomplicated cases a nurse or other technician is capable of giving an anesthetic without harm to the patient. Many surgical emergencies are unforeseen, however, and it is in such cases that the advisability of having a qualified physician to administer the anesthetic is seen. How many delicate cardiac cases owe their successful outcome as much to the skill and watchfulness of the anesthetist as the ability of the operator!

"The chief objection raised against the suggested bill was that it would make it very difficult to procure an anesthetist for service emergencies at unusual times. Where a proper anesthesia staff is organized, this should be no more difficult than to obtain the services of a surgeon or medical man. First of all, the house men should be trained in anesthesia as they are in other specialties.—*New York Times.*

MEDICAL RESERVE OFFICERS ATTEND MEDICAL FIELD SERVICE SCHOOL, CARLISLE, PA.

It is the policy of the Surgeon General of the Army to take every advantage of the educational facilities which the Army affords to familiarize medical members of the Officers Reserve Corps with their prospective duties in case of war. An innovation this year is the course for commanding officers and executives of Reserve hospital units to be given at the Medical Field Service School, Carlisle, Pa., for the 15-day period September 12-26. It is announced that this instruction will concern itself with such important functions as mobilization, administration, hospitalization and evacuation. Since the entire Eastern section of the country will be represented an excellent opportunity is presented for mutual contact and interchange of views amongst a large number of officers.

Reserve officers attending the course are placed on active duty in the grades which they severally hold in the Army of the United States. Of course this service is entirely voluntary. The healthy state of the Medical Reserve Corps in New England is attested by the fact that the following named representative medical men have applied for this course and have again donned the olive drab and gone to Carlisle:

Colonel Thomas J. Burrage, Med-Res., 139 Park Street, Portland, Me.

Colonel William H. Robey, Jr., Med-Res., 202 Commonwealth Avenue, Boston, Mass.

Colonel David D. Scannell, Med-Res., 489 Walnut Avenue, Jamaica Plain, Mass.

Lieutenant Colonel Miles D. Chisholm, Med-Res., 9 Conner Avenue, Westfield, Mass.

Lieutenant Colonel Joseph F. Hawkins, Med-Res., 197 Waterman Street, Providence, R. I.

Lieutenant Colonel Gaspare E. Lentine, Med-Res., 31 Waldeck Street, Dorchester, Mass.

Major Charles L. Judkins, Med-Res., 5 Valley Road, Swampscott, Mass.

Major William D. McFee, Med-Res., 53 White Street, Haverhill, Mass.

Major Edward F. Murray, Med-Res., 57 Pearl Street, Burlington, Vt.

Lieutenant Colonel John F. Hackett, Med-Res., 111 West Main Street, Waterbury, Conn.

Lieutenant Colonel Charles H. Holt, Med-Res., 48 Grove Street, Pawtucket, R. I.

Lieutenant Colonel James H. Means, Med-Res., 15 Chestnut Street, Boston, Mass.

Lieutenant Colonel George C. Wilkins, Med-Res., 814 Elm Street, Manchester, N. H.

Lieutenant Colonel Charles F. Gormly, Med-Res., 221 Thayer Street, Providence, R. I.

Major Louis B. Hayden, Med-Res., 79 Court Street, Plymouth, Mass.

Major George A. MacIver, Med-Res., Massachusetts General Hospital, Boston, Mass.

Major Roscoe H. Philbrick, Med-Res., Main Street, East Northfield, Mass.

Major Henry E. Pick, Med-Res., United States Veterans' Hospital, Rutland Heights, Mass.

Major Oliver L. Stringfield, Med-Res., 833 Hope Street, Springdale, Conn.

Major John H. Wyman, Med-Res., 3 Sanford Street, Medway, Mass.

Captain Edmund J. O'Shaughnessy, Med-Res., 29 Cherry Street, New Canaan, Conn.

Major Hollis G. Batchelder, Med-Res., 30 Court Street, Dedham, Mass.

Captain Charles H. Dabbs, MA-Res., 1266 Boulevard, New Haven, Conn.

Case Records of the Massachusetts General Hospital

ANTE-MORTEM AND POST-MORTEM RECORDS AS USED IN WEEKLY CLINICO-PATHOLOGICAL EXERCISES

EDITED BY
RICHARD C. CABOT, M.D., AND HUGH CABOT, M.D.
F. M. PAINTER, A.B., ASSISTANT EDITOR

CASE 12391

CARDIAC AND EXTRACARDIAC RESULTS OF A RHEUMATIC INFECTION

MEDICAL DEPARTMENT

A married English-American woman forty years old was admitted January 18 for the first time complaining of precordial pain, palpitation, dyspnea, weakness and edema. Her mother died of paralysis, her father of neuritis and senility with fluid about the heart, one brother of dropsy and inability to walk, another brother of heart disease, an aunt of tuberculosis. Ten brothers and sisters died in infancy. One brother and one sister had chorea. The patient had not been healthy. At six years she had pneumonia. From the age of fifteen to eighteen she had severe anemia with a greenish tinge, and severe cough. After paroxysms of coughing she usually found that she had been incontinent of urine. She had edema. She had been married fifteen years and had had five miscarriages and twelve years ago a tubular pregnancy with operation. She occasionally had a mild sensation of dizziness. She had a severe attack of influenza during an epidemic. She had occasional sore throat. From time to time she had indigestion, particularly after eating starchy foods. She occasionally had griping pains in the abdomen. She occasionally passed blood-streaked hard stools. At labor she usually had hemorrhoids. During the past few months she had had ringing in the ears. Upon her arrival in America, four months ago, she had diarrhea for a week, with severe pains in the stomach. Ten days before admission she again had slight diarrhea. A year before admission she had foul yellowish discharge from the vagina, clearing up after several months of douches with lysoform. At present her catamenia came every three weeks and were abnormally profuse.

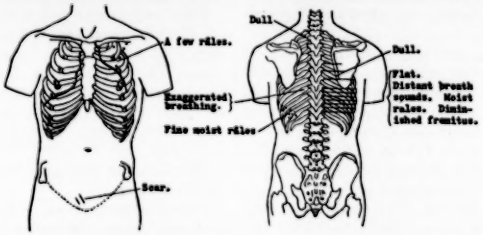
At six years she had rheumatic fever and at thirteen chorea. At the time of her anemia she was very dyspneic and had edema. After her marriage the anemia was much improved, and although she led a very strenuous life she had no more trouble with dyspnea until four years ago. Then she awoke in the night with a smothering sensation, gasping for breath. Four months after this she began to have occasional severe headaches accompanied by poor vision and spots before her eyes. These continued for six months. Then she had another severe night attack of dyspnea with a loud rattle in her throat. She could breathe in but had great difficulty in expiration. At the same time she had marked weakness of the right arm and the right side of the face. She could not speak or write. Her speech returned in five hours and the full strength of the arm in fourteen hours. After this she was up and about the house, but was dyspneic on exertion and had to sleep almost sitting up in bed. Her abdomen became greatly enlarged. After three weeks in a hospital in Vienna she felt like a new person. The edema had disappeared and she could sleep flat. For three or four years she had had palpitation at times. The January before admission she had severe pains in the lower abdomen, profuse leucorrhea, and much vomiting due she thought to digitalis. She was given electrical treatment which caused vaginal bleeding for two months. She then had dyspnea and edema, for which she entered a hospital again. She left in April greatly improved. Five months before admission she began to have attacks of sharp pain in both sides of the chest posteriorly, worse on breathing, the last attack five weeks before admission. She was then very well until she came to America in September, four months before admission. From three months until one month ago she urinated once at night and had marked burning and difficulty in starting the stream. She was kept in bed a week and had no more trouble until the middle of December, when her abdomen began to enlarge gradually. December 23 she again became dyspneic and had some edema of the feet. A week before admission she became very dyspneic and had to stay propped up all the time. She had pain about the chest, burning of the back and edema of the abdomen and neck. She had been under medical care but had not taken her own digitalis for a week. During the four years of her illness she had gained weight.

Examination shows a well nourished woman sitting propped up in bed, dyspneic, with the mitral facies. Carious teeth. Pyorrhea. Throat slightly reddened. Lung signs as shown in the diagram. Apex impulse of the heart felt 11 centimeters from midline in the sixth interspace coinciding with left border of dullness, 3 centimeters outside midclavicular line. Supracardiac dullness 7 centimeters, right border of dullness 4 centimeters at the cardial hepatic angle, the only place where it was made out. Action absolutely irregular, rate 130 at apex, 100 at wrist. Occasional periods of very rapid rate, 180 or more. At these times the rhythm seemed more regular. A harsh systolic murmur at the apex, transmitted to the axilla. Third

sound heard distinctly to the left of the sternal border in the fourth and fifth space. No diastolic murmur out definitely. Pulmonic second sound greatly accentuated and reduplicated. Mitral first sound accentuated, mitral second reduplicated. Blood pressure 140/80—150/85—110/80. Electrocardiogram: auricular fibrillation, rate 70—90, low T_2 wave, slight widening Q R-S. Abdomen: dullness, voluntary spasm, tenderness below the costal margin. Liver edge not felt. Pigmented scars on legs. Left pupil reacted sluggishly, right not at all. Corneal scars. Knee jerks and ankle jerks normal.

Under digitalis and morphia the heart rate dropped to normal by the night of January 19. At the apex there was a definite late diastolic murmur. There was fluid at the right base; not enough to justify tapping. The liver was thought to be down almost to the umbilicus, but the edge could not be definitely felt. The next day she was still more comfortable. The morning of the 21st however she had an attack of rapid heart action after which she looked much worse. By the 23rd she was well digitalized. The signs at the right base had cleared up considerably.

January 27 there was flowing again. Pelvic



Urine cloudy at two of seven examinations, specific gravity 1.027 to 1.018, a trace to a very slight trace of albumin at five examinations, leucocytes at all, including two catheter specimens, one of which showed an occasional red cell and granular casts. Another specimen was loaded with red cells, and two others showed 4-40 reds per high power field. Amount of urine normal except for one diuresis, 76 ounces, February 11. Renal function 25-30 per cent. Blood: leucocytes 7,200 to 16,000, polynuclears 72 per cent., reds 4,200,000, smear normal. Two Wassermanns negative. Non-protein nitrogen 35 milligrams.

X-rays: Marked increase in shadows of both lungs. Outline of the diaphragm indistinct on both sides, particularly on the right. Definite increase in size of heart shadow, with increased prominence in the region of the left auricle and the pulmonary artery. . . Retained tooth fragments and an area of absorption in lower molar regions. Sinuses appeared negative. . . Anterior surface of the tibiae very irregular. Some thickening of the cortex, with narrowing of the medullary canal.

Consultation, syphilologist: "The history is strongly suggestive of lues. The tibiae are rough. The clinical signs plus the X-ray make a positive diagnosis plausible. Mercury in guarded amounts is the best treatment."

Temperature at entrance 99.9°, afterwards 97° to 98.4° until the attack of tonsillitis in February when it ranged from 98.4° to 103.4°. Apex rose 120 to 58, pulse deficit 30 to 3. Respirations at entrance 40, afterwards 15 to 29.

examination showed a large, eroded and freely movable cervix. The fundus was indistinctly felt two or three inches above the symphysis. She gave a history of profuse flowing with clots every two or three weeks for four months, and similar trouble for two months the year before. A surgical consultant found no marked enlargement or deformity of the uterus. X-ray treatment was given.

February 4 she was suddenly seized with severe pain in the right costovertebral angle radiating to the right lower quadrant and symphysis, accompanied by some vomiting. Examination of the abdomen was negative. There was however some costovertebral tenderness on the right. Examination of a voided specimen of urine showed occasional red blood cells and a few leucocytes. The heart rate was 66. Two injections of morphia gr. 1/6 were necessary to give relief, and even then she did not go to sleep. She was not free from pain until the night of February 6. X-ray of the genito-urinary tract showed no shadows which could be interpreted as stone. The kidney outlines were not visible.

February 7 she had acute follicular tonsillitis. Two throat cultures were negative for diphtheria bacilli and showed streptococci. February 11 she had considerable abdominal pain probably due to distension. February 16 the acute symptoms had cleared up and she was more comfortable. At the time of her discharge, February 21, she was quite comfortable, and had no fluid in the chest, extremities or abdomen. The heart rate was slow.

March 12, nineteen days after her discharge, she reentered the hospital with an acute upset ushered in by a severe chill and vomiting followed by diarrhea and arthritic manifestations.

Examination on admission showed the right knee moderately swollen and tender, containing some fluid but not red or hot. The heart examination was essentially the same as at the first admission except for a rate of 40. The chest was clear at entrance, but during the stay in the hospital she developed at the left base fine and medium crackling râles and slight bronchial breathing, and pain in the left shoulder region. This was followed by a rub below the right scapula, where same fluid appeared later. After the knee condition cleared up wrist involvement followed.

At no time during this stay was the leucocyte count elevated. The red cell count was 4,200,000 at admission, 4,500,000 at discharge. A blood culture was negative. A right chest tap gave 45 c.c. of blood tinged fluid, specific gravity 1.013, cell count before taking 6,480, lymphocytes 88 per cent., polynuclears 6 per cent. The urine was essentially negative.

There were three periods of irregular elevated temperature ranging from 103.8° to 99.6°, the first corresponding to the pleuritic symptoms, the second to the wrist involvement, the third corresponding to neither of these complications, but apparently acute infection.

May 2 she was discharged.

June 2 she entered the hospital for the third time. The evening before this readmission while vomiting after rapid digitalization her face and her right arm became numb. Later the arm hung limp and her speech became thick. Her face and speech cleared up in a few minutes. The arm however was still paralyzed when she entered the hospital, and she also had rheumatic pains in the joints of the hands and the right shoulder.

Examination showed cyanosis and a suggestion of early clubbing of the fingers. Otherwise it was essentially as before.

The blood, urine, and a blood culture were negative. Electrocardiogram showed auricular fibrillation, ventricular rate 40, diphasic T_2.

During this stay she had tonsillectomy under ether at the Eye and Ear Infirmary and went through the operation remarkably well.

July 6 she was discharged to another hospital.

She apparently was fairly well after her discharge until November 1. Then she began to be troubled at times with nausea and vomiting and was in bed a good deal. Two weeks before her fourth admission, January 26, she began to have increased dyspnea, orthopnea, swelling of the ankles, nausea and vomiting.

On examination she was cyanotic and very dyspneic. The lungs showed dullness and moist râles at the left base posteriorly, flatness with diminished tactile fremitus and breath sounds at the right base. The heart findings were essentially as before except that no diastolic murmur was heard. There were no acute joints.

The leucocyte count was 8,500 to 15,000, the red count 6,100,000 to 5,200,000, the hemoglobin 80 to 75 per cent. The urine showed the slightest possible trace to a large trace of albumin, specific gravity 1.010 to 1.030. Sediment; no red cells to many, a few to many leucocytes, no casts to occasional hyalin and granular casts (no catheter specimen). Renal function 40 per cent. Right chest tap 1150 c.c. of blood tinged fluid, specific gravity 1.008.

The temperature was only slightly elevated at any time during the stay.

February 26 she was discharged.

After leaving the hospital she was up and about, at first in her room, later going downstairs. March 4 after dinner she felt chilly. Suddenly her left side became paralyzed. After an injection by a physician she came out of the initial stupor and confusion, but soon relapsed into coma.

March 5 she was admitted to the hospital for the fifth time.

Examination showed her unconscious, noisy, uttering inarticulate sounds. Left hemiplegia. Auricular fibrillation, pulse deficit 90 to 60. A few râles at the left base.

The temperature was 98.6° to 106.3°. The apex pulse was 66 to 123, pulse deficit 33 to 5. The respirations were 23 to 48. Blood pressure 158/120.

The patient remained unconscious. The lungs filled with moist râles. Very loud breathing was heard below the right clavicle. March 11 she died.

Discussion

By Richard C. Cabot, M.D.

Notes on the History

1. This patient has a pretty full assortment of cardiac symptoms.

2. I should say that her father died of heart trouble, and the chances are her brother died of heart trouble too.

3. At fifteen it sounds like the old-fashioned chlorosis, one of the few diseases which we know have died. Ordinarily diseases do not die unless we find their cause and root it out, as in the case of some of the infections. But chlorosis seems to have died.

4. This incontinence of urine has no special significance, of course. It merely means that coughing was severe.

5. We do not know just when she had edema; probably in her youth.

6. The blood-streaked stools were presumably due to hemorrhoids.

7. In the past history I find very little in the way of suspicions to carry forward with me.

8. We have a well marked rheumatic history which makes us think of mitral valve disease, and less commonly of aortic valve disease.

9. This is not the usual type of cardiac dyspnea. Ordinarily there is just as much trouble one way as the other. This is more the type that often receives the name of asthma, and sometimes, if there are other cardiac symptoms, of cardiac asthma.

10. She evidently had a cerebral embolism, probably coming from the left auricular appendage, which is the commonest place for clots. She has the disease which ordinarily leads to mitral stenosis, and we have good reason to guess that that was a cerebral embolism from a piece of that clot.

11. This is a straight cardiac history except for one or two things which suggest kidney. The headache and cerebral symptoms were a little more like kidney than like heart, aside from those that go with embolism.

NOTES ON THE PHYSICAL EXAMINATION

1. "Mitral facies" means a combination of cyanosis and pallor.

2. In the diagram I understand everything except the dullness at the top, and that I am dubious about. In the front the râles are on the same side where we get dullness behind, and may be due to tuberculosis, but there is nothing else to suggest it.

3. There is a big heart. It is not obvious from these measurements that it is more enlarged transversely than downward. It is enlarged both ways, as it usually is. They were looking hard for a diastolic murmur.

4. The blood pressure really is of no importance in a case like this, because different beats are of different sizes. The diastolic does not vary much.

5. The corneal scars suggest the reason the pupil does not react. There may be some old iritis as well as some conjunctivitis.

6. These urinary reports all mean normal kidneys except for passive congestion and possibly an infarct. The blood in a catheter specimen may be due to an infarct. It is just the sort of case when infarcts would be expected. The non-protein nitrogen is normal.

7. There is a slight anemia.

8. The X-ray shows a chest largely waterlogged. We cannot make out the diaphragm. There is much fluid on the right, less on the left. That is the rule: when we have dropsy in the chest it is more on the right. There is more density in the upper lung fields, but the apices are clear. That is important because we have a suspicion of a tuberculous process. But there is nothing here.

Here is a seven-foot plate of the heart. The heart shadow is strikingly wide across the region of the auricles, nearly nine centimeters, which supports the guess already made of a mitral disease. The total width of the heart is certainly increased, both to right and left.

The findings in the tibiae make us suspicious of syphilis. The other plates were taken in relation to pyogenic foci.

9. This is often the history,—that when the heart is going fast we cannot hear much of anything. We get it slowed down with digitalis and rest and then the murmurs come out.

10. X-ray treatment was given for the uterine hemorrhage. It is good treatment, and very effective in many cases.

11. Under these conditions the attack of February 4 might well be a renal infarct. Sudden pain anywhere in that region suggests it.

12. Every now and then a person in a ward where there is no opportunity for infection, with rheumatism sometimes, with heart disease sometimes, begins to have tonsillitis. It makes me feel that tonsillitis and rheumatism do not cause heart disease, but that they are all of them branches off the same trunk, the results of the same cause.

In her first entry we carried her through an attack of broken compensation, presumably with an acute infection, and then she was discharged.

At the second entry she has rheumatism, which she had not had before since she was a child.

13. A PHYSICIAN: How do you explain that drop in the heart rate?

DR. CABOT: Two explanations come to mind: first that she has taken digitalis enough to drive it down, which I do not think is likely; and secondly that she has a real digitalis heart block. In a woman of this age we need not suppose any lesion in the bundle of His.

14. It is this recurrence of the acute cause that makes us have a bad prognosis in heart cases. They get along well enough so far as the mechanical condition of the valve is concerned, if they do not have a recurrence. But this recurrence means increase in the condition on the valve lesion and bad prognosis, usually.

She was discharged probably to a chronic hospital, because she could not be kept here.

At her third admission there were, as before, more findings on the right than on the left.

15. The final blood pressure was due presumably to the infarct. Increased pressure in the brain will give it.

DIFFERENTIAL DIAGNOSIS

This is a very typical history, I should say, of a rheumatic lesion which began, I suppose, when she was six and terminated when she was forty-one. It began with rheumatism. She later had chorea. Here she had tonsillitis without any fresh outside exposure. She also had another attack of rheumatism here. She had a heart characteristic of mitral disease, at no time characteristic of aortic disease, although that may be there. And she had several attacks, at

least three I should say, of cerebral embolism, and one attack suggesting infarction of the kidney.

Did she ever have an acute endocarditis? It is perfectly possible that with one of these joint troubles or at other times she had an acute process there, and it is perfectly possible that it will be there at the end, and Dr. Richardson will show it. But we do not need to suppose it. She could have all these symptoms simply from bland non-septic emboli in the left auricle, with pieces broken off and going to the kidney or the brain. She ought to have mitral stenosis, because that is the commonest rheumatic lesion and because the murmurs heard are consistent with that, although a diastolic was not heard. Will she also have an aortic lesion? There is no good evidence of it. It is perfectly possible, but it seems to me that with the signs given it is improbable. What about a tricuspid lesion? We never diagnose that. It may be there, but we cannot say so.

The pericardium may be involved. There may be a chronic pericarditis here. We do not get signs. All we can do is to suggest it.

A PHYSICIAN: Would the shape of the heart suggest it?

DR. CABOT: No, it would not to me. It is a normal shape I should say.

We ought to commit ourselves as to what Dr. Richardson is to say about the head. He ought to say right-sided cerebral embolism with softening. There might be signs of old infarcts in one or both kidneys and in the spleen. Passive congestion of the lungs, liver, and all the organs is all I see that we should mention here. Unless there is complicating acute endocarditis there should be no bacteria in the findings at necropsy.

A PHYSICIAN: Assuming the presence of mitral stenosis, is there any way to differentiate emboli from the valves from those found in the auricular appendix?

DR. CABOT: Thrombi on the valves usually go with a streptococcus viridans process, and they usually give rise to some septic manifestations such as purpura, such as abscess in the lung or elsewhere, or to a septic fever. She has not had either. That does not prove that she did not have an acute streptococcus endocarditis on top of the old one, but it makes us unable to say so and makes it rather wiser to say that she did not.

A PHYSICIAN: May we see the picture of the tibiae?

DR. CABOT: I imagine the X-ray expert would say about what the text says about those shins.

A PHYSICIAN: How do you explain the lesion on the tibiae?

DR. CABOT: The X-ray man seems to think it is due to syphilis. That means that at some time this man has had syphilis. It does not mean that there is any syphilis now or that we shall find any postmortem.

A PHYSICIAN: Doesn't streptococcus periostitis produce a similar lesion?

DR. CABOT: Not so far as I know. There is a possibility that she had a hereditary syphilis. It is a rare thing at this age. It is possible but unlikely. Out of thousands of cases we see perhaps one that lives to forty with hereditary syphilis. I do not believe the necropsy will show any evidence of syphilis. I believe that partly on the history, which aside from the shin does not give any evidence, and partly because our necropsies here very seldom do show any evidence. Syphilitic lesions, aside from the heart, testis and liver, are very rarely found in this clinic. So I do not make that diagnosis nowadays.

A PHYSICIAN: How do you account for the lesions in the eye?

DR. CABOT: Those might be rheumatic. Rheumatic iritis is common and would account for all of this.

A PHYSICIAN: She had opacity of the cornea.

DR. CABOT: Yes. That may be syphilitic of course. I think it was probably interpreted so by the man who was there, because she had syphilitic treatment.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Rheumatic heart disease with mitral stenosis.
Auricular fibrillation.
Cerebral embolism.
Left hemiplegia.

DR. RICHARD C. CABOT'S DIAGNOSIS

Chronic endocarditis, mitral stenosis.
Cerebral embolism, right-sided, with softening.
Hypertrophy and dilatation of the heart.
Possibly old infarcts of the kidneys and the spleen.
Passive congestion, general.

ANATOMICAL DIAGNOSIS

1. *Primary fatal lesion*

Chronic endocarditis of the mitral valve, stenosis.

2. *Secondary or terminal lesions*

Hypertrophy and dilatation of the heart.
Slight arteriosclerosis.
Chronic passive congestion.
Infarcts of the spleen and kidneys.
Infarcts of the lungs.
Large area of anemic infarction and softening of the right cerebral hemisphere.
Edema piae.

3. *Historical landmarks*

Chronic pleuritis.
Cholelithiasis.
Scar of old operation wound.

DR. RICHARDSON: The head examination showed the vessels of Willis to be negative except that the right middle cerebral artery, leading to the margin of a very extensive area of anemic infarction on the right, was engorged; but unfortunately just about at the point where we might expect to find an embolus the vessel was disintegrated and lost in an infarcted area. This area of infarction was quite extensive. Looking down on the brain from above, it extended from the anterior border of the caudate nucleus back to the anterior portion of the occipital lobe and out nearly to the cortex. The pia was rather wet. The left side of the brain was negative, as were the pons, medulla and cerebellum.

Trunk. There was an old operation scar 8 centimeters long just above the pubes. The peritoneal cavity and appendix were negative, as were the uterus and adnexa. The gastro-intestinal tract showed a rather slight amount of passive congestion. The liver was at the costal border. The diaphragm on the right was at the fourth interspace, on the left at the sixth rib.

In the right pleural cavity there was 150 cubic centimeters of transudate. The left was obliterated by old adhesions. There were some old adhesions on the right. The lungs showed passive congestion and a small infarct in the region of the lower lobe on each side.

The pericardium was negative. The heart weighed 482 grams. That is considerably enlarged for her. The myocardium generally was thick, four millimeters on the right and fourteen millimeters on the left. The cavities showed slight dilatation on the left, moderate on the right. The mitral measurement was six centimeters; stenosis. The curtain showed frank chronic endocarditis, that is marked fibrosis, extending along the curtain of the valve, as a thick broad band up to four millimeters thick in places, with shortening and thickening of the chordae tendineae and consequent decrease in the circumference of the valve. On the superior surface of this valve, up toward the auricular appendix but not in it, there was a small irregular crevice with a rough reddish base. At the time there was no definite evidence of a thrombus there, but we have to account for that reddish roughened base, and it might very well be that it was the region of attachment of thrombotic material from which bits had gone up into the right brain and down into the spleen and kidneys, because we found infarcts in these organs.

The aortic valve measured six and a half centimeters, the tricuspid twelve and a half, the pulmonary seven and a half. They were negative. A case then of pure mitral disease. The coronaries were free. There was a slight amount of fibrous sclerosis of the aorta scattered along it here and there. The great branches were negative.

The liver weighed 1160 grams,—chronic passive congestion. Extending from its left lobe down to the spleen there were some old adhesions, with those at the splenic end attached to the outer surface of an infarct. In this particular case, and at this time, the condition was simply chronic peritonitis, but at some previous time there was probably some localized acute peritonitis there.

In the gall-bladder we found one stone eleven millimeters in diameter. The mucosa was negative, the bile-ducts free. The spleen weighed 137 grams and showed well marked chronic passive congestion, and in the region of the lower pole a large infarct, three by two and a half centimeters, its outer surface bound by adhesions to the left lobe of the liver as mentioned. The kidneys weighed 285 grams. They showed of course some passive congestion, but other than for that and a few small infarcts they were negative macroscopically and microscopically.

The heart blood yielded no growth. The heart was of pretty good size for a pure mitral.

DR. CABOT: Yes, but we have nothing else.

CASE 12392

RECURRENT DYSPHAGIA

MEDICAL AND LARYNGOLOGICAL DEPARTMENTS

A seventeen-year-old boy was seen privately, consulting because of occasional weak spells, nervousness and difficulty at irregular intervals with deglutition. He stated that when attending a class dinner, when dining with friends or when teased by his brother about his affliction he not only could not complete the swallowing process but would of necessity leave the table, and, as he stated, vomit his food. He was a neurotic youth with mottled cyanotic clammy hands, blushing easily, sweating easily and ill at ease. He stated that he seldom had trouble in the morning, occasionally at noon and generally at night. He had more trouble when especially tired. He was especially anxious because he has no control over the regurgitation. In taking his history the following report was obtained. This history includes the subsequent experience and treatment following the above consultation.

A six-year-old boy came to the Out-Patient Department for relief of vomiting November 28, thirteen years before his admission to the Eye and Ear Infirmary. His father died of "fatty degeneration of the heart and kidney disease." One grandmother had syphilis. The child was treated for syphilis in infancy. He was very difficult to feed, "vomiting" frequently on many various formulae. He had measles at eleven months, followed by scurvy. He had always vomited immediately on taking solid food,

and had lived on liquids and very soft solids. The regurgitated food was not sour, but was mixed with mucus. He occasionally regurgitated liquids. He did much better while taking chloretone. He never complained of pain until two weeks before coming to the Out-Patient Department, when he had soreness in the epigastrium an hour after breakfast lasting until two days before the visit. For three weeks he had had unproductive cough.

Physical examination. Well nourished. Head large. Forehead bulging. Eyes normal. Middle incisors saw-edged. Cervical, axillary and inguinal glands enlarged. Heart, lungs and abdomen normal. Knee-jerks lively. Von Pirquet negative. Wassermann tests on the mother and boy were negative.

X-ray. Bismuth in liquids passed to the stomach without any apparent obstruction. Solids were partially obstructed opposite the articulation of the first and the second segment of sternum. There was no marked dilatation of the esophagus above the point of obstruction. Examination of the chest before giving bismuth showed some thickening in the posterior mediastinum opposite the lung roots.

Non-malignant constriction of the esophagus about four centimeters above the diaphragm. The lumen at this point is about one-third the normal diameter.

December 26 he was admitted to the wards. Under ether a small Coolidge esophagoscope was passed without difficulty to nine and a half inches below the teeth. At this point the scope turned to the right in order to follow the opening of the esophagus. After this curve was passed the stomach could be seen straight ahead. Impression, cardiospasm. Bougies up to size thirty-six were passed into the stomach without difficulty.

December 30 he reported at the Out-Patient Department that he had been very much improved since the esophagoscopy. He could eat things like bread and meat, which he had been unable to swallow before. January 8 he still was better than before the operation, though not so well as at the last visit. January 20 the dysphagia was about the same as before the operation. He still had some cough, mostly at night and in the morning. The tonsils were enlarged and reddened.

January 23 he was readmitted to the wards. It was noted that his inability to swallow varied considerably. At times he could eat bread and at other times had trouble with water. Meat was always regurgitated. The food returned unchanged, as though it had never entered his stomach; the amount was never over a tablespoonful.

Examination was as before. The upper incisors were broad but very slightly triangular, had rather poor enamel and slight notches in the biting edges. A slight systolic murmur was heard best along the left sternal border. The stools and urine were not remarkable.

Dilatation of the esophagus was done. A number thirty-six bougie was passed afterwards without any difficulty.

February 3 he was able to eat everything and had had no vomiting since the last note. February 21 he was doing well, but had one or two spasms a week.

June 30 he returned to the Out-Patient Clinic complaining of regurgitation after every meal.

July 8 he was readmitted to the wards. Under ether the esophagus was dilated sufficiently to allow a number thirty-six French bougie to pass easily.

July 13 he was eating soft solids and had had no vomiting since the dilatation. July 20 a number twenty-eight bougie passed with ease.

A year later his mother wrote that he had occasional attacks of regurgitation, but that he had made marked improvement during the year and was gaining weight.

After the office visit mention at the beginning of the history he was referred to the Eye and Ear Infirmary for X-ray examination. An abstruction was found at the middle third of the esophagus, thought probably to be a web.

January 1 he was admitted to the wards of the Eye and Ear Infirmary. In giving his history he reported that the trouble varied. Occassionally it was thirty-six hours before he could get food into his stomach. Then he would have no trouble for perhaps three days.

January 14 operation was done. Under ether an esophagoscope was passed. At fourteen inches from the teeth an ulcerated area was found above and below the tube, and not bleeding. The operator questioned whether this was true ulcer or abrasion from the suction tube. The latter was smooth ended and had been carefully greased. Bougies up to number thirty-

two French were passed through the area without difficulty.

January 16 the patient was discharged in improved condition.

January 30, two weeks later, X-ray examination showed very much less delay at the point of stricture.

Discussion

BY B. THURBER GUILD, M.D.

I have not tried to make a puzzle picture of this case, but to get some information rather than to give it.

This boy was obviously nervous. He was ill at ease in the office, had the mottled, cyanotic hands of the nervous person, and he said that his attacks were more apt to come when he was self-conscious. My impression was that it might be what I then accepted as a possible entity, cardiospasm.

In getting his history I found that he had been very hard to feed as an infant, had been on several formulae and under several different men's care.

Regurgitated food mixed with mucus I think is an important diagnostic point.

X-ray gave no key whatever to the situation.

The esophagus was dilated at the first visit, again at the end of a month, again at the end of six months, I think again at the end of a year, and again at the end of nine and a half years.

Obstruction was found at the "middle third" of the esophagus. I think that is rather an indefinite location. We do not know just where it is, and it makes a difference.

I do not know what they mean by "above and below the tube" unless they mean that in the horizontal position of the patient there was ulceration above and below it.

The interest in this case is enhanced by the ability to carry back a history to infancy, when the trouble was thought to be purely a case of so-called difficult feeding. The progress to recognizable pathology was slow. Not until the age of seventeen was a definite stricture or web formation demonstrated. It is also interesting to see the increasingly satisfactory results of bougies. His first relief was for one month, then for six months he was practically free from trouble, then for one year, and then for nine and a half years he felt no necessity for interference.

The questions raised in my mind are these: Is there ever a purely functional cardiospasm? Has this case a syphilitic background in fact as well as historically? Could this be a case of peptic ulcer of the esophagus, causing cicatricial formation?

In regard to pure cardiospasm I feel now that as in hysteria and neuroses, we are only temporizing with such a diagnosis. Thirteen years ago Rotch in an article on "Types of Occlusion of the Esophagus in Early Life" stated: "It is possible that pure spasms of the esophagus are mostly congenital and are located in the brain."

I can throw no light on the syphilitic aspect of the case and would like to hear something about it. Syphilitic lesions are probably higher than this. I should expect concomitant symptoms.

In regard to peptic ulcer the lesion seems too high. It would have to be near the cardia. The ulcerated area is reported as being fourteen inches from the teeth. If not traumatic, what else would cause ulceration?

Tileston reported in 1906 an analysis of forty-four cases of ulcer of the esophagus ranging in age from infancy to sixty-six years. This patient complained of pain at one time for a short period. I shuld expect if this were peptic ulcer that the vomitus would be acid unless the ulcer early formed the web which subsequently formed a barrier between the bolus of food and the gastric juice. There must have been early pathology which caused definite and progressive symptoms but which were negative to X-ray and operator until the patient was seventeen years old.

If it were a congenital defect I should expect the X-ray to show something when first used in childhood; it was not a neurosis; therefore it is suggestive of some progressive ulcerative or infectious process.

Opposing surfaces of ulcerated or inflamed areas in this structure where there would be periods of quiet and thus a chance of adherence of the parts could in my conception be a cause of web formation or stricture. The causes of such an ulceration or inflammation might be syphilis, peptic ulcer, chemical trauma, or as has been suggested in the literature, it might be secondary to one of several acute infectious diseases,—diphtheria, scarlet fever, etc. It is interesting that in most esophageal occlusion cases the patients are noted as being especially nervous. "Nervousness might precipitate more frequent attacks, but I believe an anatomical cause must coexist."

Subsequent History. July 14, 1926, at age nineteen, a year and a half after the last operation, the patient reports a mild transitory attack of spasm occurring perhaps every two or three weeks. A complete examination of him shows his nervous system negative as to reflexes. He has migraine headaches every one or two weeks. His Wassermann is negative. He still shows the systolic murmur best heard at the third left interspace; no enlargement. Pulse rate 100, increasing on exercise but quieting normally to the initial rate. Blood pressure 136/40.

Sometimes by waiting patiently he will be able to complete deglutition. At other times—and he always knows when—he must disgorge the bolus of food, most frequently meat, and

then resume his meal without further difficulty. He says if he does not get worse he will consider himself cured—quite a logical deduction, though somewhat ambiguous!

I think a case of this kind was reported in these Case Records some time ago.* My interest was mainly in being able to carry the history back so far.

A PHYSICIAN: What do you mean by a web?

DR. GUILD: A membranous structure like a diaphragm stretching across the lumen of the esophagus either with a central opening, or crescentic in shape with the opening near a side of the wall. I imagine that in web formation a bolus of food comes down and by irritation of the pathological portion causes constriction, or spasm. He cannot voluntarily relax it, and if it persists he is obliged to disgorge.

A PHYSICIAN: A web of that sort would naturally be ruptured by the passage of the bougies?

DR. GUILD: Possibly not ruptured so much as stretched when they are passed. Dr. Mosher frequently cuts them, I believe. The ulceration was found when the esophagoscope was passed —before operating.

A PHYSICIAN: The passing of the bougies would not have caused ulceration?

DR. GUILD: No. I understand that when the scope was first put in the ulceration was found. There was no trauma before the ulceration was found. It was not bleeding, so it was not an acute thing at that time.

DR. LANE: I was interested in the question of syphilis, but my impression is that while syphilis can occur anywhere it is probably very infrequent in congenital cases and in the esophagus. Of course a few stomach cases have been reported and a few tertiary lesions in adults, but offhand I cannot recall any gummatous or ulcerative lesion of the esophagus itself. Treatment for syphilis in infancy I should say does not necessarily mean syphilis in the child, even with the history of syphilis in the grandmother. The lack of confirmative symptoms would to my mind be rather against its being syphilis.

DR. GUILD: I do not think he had any congenital symptoms. He had some symptoms of rickets.

A PHYSICIAN: I should like to ask about that mottling. Did that extend to the rest of the body?

DR. GUILD: No, except that he always had cold perspiring feet. He is of the type, frequently women, who when emotionally aroused have mottling over the upper part of the chest and neck.

A PHYSICIAN: Williams in New York last year or the year before reported a number of these permanent mottlings of the skin which had lasted for a long while under observation.

*Case 12091, March 4, 1926.

DR. GUILD: My impression was that this was a vasomotor disturbance.

A PHYSICIAN: It usually is, but these are the first I have seen reported as permanent.

DR. GUILD: Should you say then that syphilis would be very unlikely? In the face of the history as to the grandmother and this ulceration without knowing any etiology, you would not be led to be suspicious?

DR. LANE: Do you know whether the grandmother had treatment?

DR. GUILD: No, I do not.

DR. LANE: With the history of syphilis in the grandmother the old question is brought up as to whether it is absolutely carried through to the third generation.

DR. GUILD: I was going to ask if syphilis, like other hereditary things, is apt to follow the law of atavism and skip one generation?

DR. LANE: There is a good deal of question in my mind as to whether it has been carried over, because of the difficulty of excluding it in the preceding generation. There have been some few cases recorded, but most authorities I believe are somewhat skeptical about it.

DIAGNOSIS

Esophageal web.

CASE 12393

A CASE SHOWING THE DANGERS OF PERSISTENT DIARRHEA NOT PROMPTLY TREATED

SURGICAL DEPARTMENT

An American farmer sixty-one years old entered February 2 complaining of "ulcers of the rectum" of six months' duration.

The November before admission he first noticed a mucous discharge from the rectum. At the same time he had sudden onset of diarrhea seven to eight times a day, generally once with a movement; at the other times he passed merely mucus. He noticed bright blood streaks daily. The diarrhea had continued with varying severity. A physician found nothing upon digital rectal examination, but on a proctoscopic examination later found a "bunch" in the rectum. Three days after this the patient passed a "bunch of grapes" about five inches long. Immediately after this he had normal bowel movements for a week without blood or mucus. Then the diarrhea recurred with bright red blood and mucus daily until admission. During the past six weeks he had had dull burning pain in the lower rectum increasing in severity. His weight had fallen from 176 pounds in the autumn to 145 pounds. He had lost some strength.

He had had measles, scarlet fever, typhoid

fever and mumps. Until recent years he used to have bad headaches.

His father died of tuberculosis, his mother of heart disease, one brother of "dropsy". His wife had never been pregnant.

Examination showed a somewhat undernourished man apparently in poor health. The skin was dry, scaly and shriveled. The teeth were very poor. There was marked pyorrhea. The apex impulse of the heart was in the fifth space 7.5 centimeters from midsternum. The percussion measurements are not recorded. The sounds were rather faint and ill defined. There was a short blowing systolic murmur. In the right epigastrium just under the costal margin a large round non-tender mass could easily be seen on inspection, descending on deep inspiration two inches. On palpation this seemed to be definitely fixed. On palpation and deep inspiration the whole mass seemed to be nodular and to be attached to the liver. Percussion above it gave flatness. There was a large right inguinal hernia which without support extended into the scrotum. On the left testicle were a number of fluctuating cystic masses. Rectal examination showed a multilobular mass across the posterior wall of the rectum involving both sides, firmly fixed and quite hard to palpation. Proctoscopy showed a sloughing, bleeding mass about three inches within the anus.

Before operation the blood and urine were normal.

X-ray showed both lung fields unusually large and bright. There were a few dense glands at the left lung root. The aortic knob was prominent. The diaphragm was a little high on the left but moved normally with respiration on both sides.

February 4 operation was done. The patient made a good operative recovery and was quite comfortable. February 14 there was a slight upper respiratory infection of the throat and fauces. The chest was clear.

February 18 a second operation was done. That day the patient was in fair condition, though pale and perspiring profusely. The pulse was rapid but of fair quality. There was moderate staining of the dressing. He was given subpectoral saline. At two a. m. the radial pulse was not obtainable. He was given 1000 cubic centimeters of five per cent. glucose intravenously and revived so that he answered questions intelligently. Before a donor for transfusion could be obtained he became unconscious, and just before the transfusion was started he died.

Discussion

BY EDWARD L. YOUNG, JR., M.D.

Any such diagnosis as this given by a patient should bring a picture before our minds not only of the actual condition mentioned but of every other possibility that might in a layman's mind be included in such a diagnosis. Here we think of ulcerative colitis and malignancy as the two diseases most likely to fit. The duration of the disease is not great if we compare it with other cases we have seen here where blood and mucus have been present for from one to two or even two and a half years. This is only three months. The description of what was found as far as it goes means a tumor and not ulceration.

The "bunch of grapes" which the patient passed might have been clots of blood or possibly bits of tumor covered with blood. If the story is true it certainly suggests that the passage was more free for a few days after the proctoscopy.

The loss of weight is consistent with either diagnosis mentioned.

The examination shows a poor surgical risk. The heart muscle is worn out and probably will not stand a great deal. The mass which is spoken of in the right epigastrium brings up the fear first that he already has metastasis in the liver. We have nothing in the history to make a gall-bladder seem probable, but that is of course possible. A somewhat enlarged lobe of the liver without obvious cause may also explain it.

The cystic masses spoken of in the left scrotum are probably spermatoceles.

Rectal examination and proctoscopy make the diagnosis certain. The only question now is the question of treatment. He is a poor surgical risk. The mass is according to this statement firmly fixed, which implies extension outside the wall of the bowel. If operation is to be done it is on the basis either that the diagnosis as to the extent of the disease can not be made accurately without laparotomy or that a palliative operation will make him more comfortable.

X-ray Interpretation

Examination shows evidence of arteriosclerosis and of emphysema of the lungs.

Dr. Young's Pre-operative Diagnosis

Carcinoma of the rectum.

Pre-operative Diagnosis February 4

Carcinoma of the rectum.
Metastatic nodules in liver.
Diverticulosis.

First Operation

Ethylene. A paramedian incision was made in the lower abdomen. The mass in the liver was found to be firm, somewhat irregular, 2½ by 2 inches in diameter, occupying the lower portion of the left lobe. The growth was found to be very firmly adherent to the left side of

the pelvis. It was determined to do a colostomy at this time and if the patient continued to have bleeding and tenesmus to do an amputation of the rectum from below later.

There were numerous diverticula along the sigmoid but no evidence of any inflammatory area.

POST-OPERATIVE NOTES

February 6 the colostomy was opened with benzine cautery. There was good escape of gas with much relief. February 12 there was a normal movement through the colostomy opening.

FURTHER DISCUSSION

The palliative operation is what was uppermost in the minds of the surgeons, as it seemed to them that the pain and the bleeding might be lessened by a colostomy, and the mass in the right epigastrium is definitely found to be a metastasis. On the basis of what the surgeon notes in the operative report we must assume that bleeding and pain continued and that the second operation was done to try to remove the bleeding, sloughing mass.

DR. YOUNG'S PRE-OPERATIVE DIAGNOSIS

Carcinoma of the rectum.

SECOND OPERATION, FEBRUARY 18

Spinal procain. The growth in the rectum was more extensive than it had been supposed to be at the previous examination. The whole retroperitoneal tissue about the rectum was infiltrated with growth, and a firm, hard mass extended into the pelvic wall on the right. The whole rectum was very firmly adherent to the pelvic wall, making it very difficult to pull down any rectum from the hollow of the sacrum. Because of this difficulty no attempt was made to go high above the growth.

PATHOLOGICAL REPORT

A section of the lower rectum. There is a deeply ulcerated growth measuring 3.5 by 5.5 centimeters, with prominent elevated margins. Its lower margin is 4 centimeters below the anal ring. Its center is deeply ulcerated, is firmly fixed to the pararectal tissue and sends a hard mass about the size of a hen's egg into it.

Microscopic examination shows adenocarcinoma with invasion of the perineural and periarterial lymph spaces of the pararectal fat.

FURTHER DISCUSSION

The operation was too much for him. He died with all the symptoms of shock. Whether there is bleeding intra-abdominally is hard to say, but a certain amount of bleeding is of course an additional factor in his death. In view of the suffering he would almost inevitably have had we cannot be entirely sorry for such an outcome in this case.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Carcinoma of the rectum.
Post-operative hemorrhage.
Operations: first stage colostomy, second stage removal of rectum and carcinoma.

DR. EDWARD L. YOUNG'S DIAGNOSIS

Carcinoma of the rectum.
Operative shock.
Hemorrhage.

ANATOMICAL DIAGNOSIS

1. *Primary fatal lesions*

(Adenocarcinoma of the rectum.)
Metastasis in the liver.

2. *Secondary or terminal lesions*

Hemorrhage into the pelvic cavity, the retroperitoneal tissues, and the lower end of the large intestine.
Anemia.
Soft hyperplastic spleen.
Moderate arteriosclerosis.

DR. RICHARDSON: The skin and mucous membranes were very pale. In the anterior abdominal wall between the umbilicus and the pubis there was a linear scar 14 centimeters long. In the central portion there was an opening three centimeters long. The sigmoid was sutured in the wall of the opening and there was a surgical opening in it. Posteriorly between the buttocks there were three operation wounds, a long sutured wound and two short ones. There was subcutaneous fat in small amount. The muscles were thin and pale. The subcutaneous tissues in the anterior thoracic wall were infiltrated with thin pale clear fluid.

Peritoneal cavity. In the pelvic region there was considerable fluid blood and blood clot.

Small intestine rather empty. Mucosa negative. Large intestine not distended. In the region of the sigmoid the intestine was sutured to the wall of the wound as mentioned. The distal portion of the intestine along a length of about 19 centimeters contained considerable blood and blood clot. The lower end of this portion of the intestine was sutured off, and in the region of the sutures within and without there was much infiltration with blood and blood clot and much infiltration of the retroperitoneal tissues in the region.

The mesenteric and retroperitoneal glands were negative.

The anterior margin of the liver on the right side was at the costal border in the right mammillary line. On the left side the anterior mar-

gin was 10½ centimeters below the costal border in the left mammillary line. The diaphragm on each side was at the fifth interspace.

The pleural cavities were dry. There were no pleural adhesions. The trachea and bronchi contained much pale froth. The bronchial glands were negative.

Lungs. The apices were negative. There were no areas of consolidation. The tissue generally was spongy and very pale. There was slight edema and some emphysema in the peripheral portions of the lungs.

The pericardium was negative. The heart weighed 272 grams. The myocardium was of good consistence and pale. The valves and cavities were negative. The aorta and great branches showed a moderate amount of fibrous sclerosis.

The liver and mass of new growth that was present weighed 1550 grams. The new growth weighed 340 grams. The surface of the right lobe of the liver was smooth. The tissue was of good consistence and pale. In the substance of this lobe, posteriorly, a short distance beneath the surface, there was a small mass of new growth tissue 1.5 centimeter by 1 centimeter. The left lobe on the anterior surface showed a boss-like mass of new growth tissue 10 centimeters in diameter. The anterior surface rounded up beneath the capsule and posteriorly the mass extended nearly through the lobe. The lower border of the mass anteriorly reached nearly to the anterior margin. In the region of the peripheral portions of the new growth the adjoining liver tissue was infiltrated with blood, and there was some hemorrhagic infiltration of the new growth tissue. The tissue of the lobe elsewhere was of good consistence and pale.

The spleen weighed 295 grams,—slightly enlarged. The surface was smooth, the tissue pale and rather soft.

The kidneys combined weighed 240 grams. The organs were frankly negative except that the tissue was quite pale.

INTRODUCTION TO THE ELEVENTH ANNUAL REPORT OF THE NATIONAL COMMITTEE FOR THE PREVENTION OF BLINDNESS, INC.

THE National Committee for the Prevention of Blindness, originally organized as a local New York State Committee through the zealous efforts of a few public-spirited individuals in 1908, took on its national character in 1915, and has grown into an agency of even international scope. It includes more than 16,000 members. Its staff has grown accordingly to twenty-one members, including a managing director, associate director, medical director, secretary, assistant secretary, membership secretary, and research secretary. In this time, also, it has been found necessary to seek larger quarters. This growth has been in proportion to the needs met. The Committee has learned that it is not only possible to prevent actual blindness, but to prevent the development of eye defects which, though not rendering individuals blind, reduce their social efficiency. The slogan may very well be not only conservation of vision, but conservation of 100 per cent vision.

It is perhaps auspicious at this time to state the position of the National Committee in the general public health movement. The following facts are indicative:

1. It is endorsed by the National Information Bureau.
2. It has co-operated in activities with the American Medical Association, the National Education Association, and the United States Public Health Service, as well as with the medical profession in general.
3. It is a member of the National Health Council.
4. It has been selected as a special member of a Committee on the Conservation of Vision of the State and Provincial Health Authorities of America, which includes Canada as well as the United States.
5. Its position in the esteem of the American Ophthalmological Society can be deduced from the fact that a resolution was passed asking the National Committee to act as a clearing house for all information on the conservation of vision.

The function of the National Committee for the Prevention of Blindness is to keep abreast of the scientific advance in ophthalmological knowledge of refraction, disease and operation; to inform the public generally in layman's language of such advance, and the ways in which such knowledge and practice may be made available to the public, together with what action, public or private, on the part of the community or private organization will provide the means for professional ophthalmological assistance.

NEW HAMPSHIRE MORTALITY STATISTICS: 1925

THE Department of Commerce announces that the 1925 death rate for New Hampshire was 1,452 per 100,000 population as compared with 1,416 in 1924. This increase in 1925 is more than accounted for by increases in the death rates from influenza (from 22 to 50 per 100,000 population), diseases of the heart (from 233 to 252), cancer (from 136 to 148), and accidental falls and automobile accidents (from 14 to 19, each).

Decreases in 1925 were shown in death rates from tuberculosis, all forms (from 74 in 1924 to 66 per 100,000 population), nephritis (from 108 to 103), measles (from 6 to 2), and whooping cough (from 10 to 6).—*Department of Commerce, Washington.*

THE BOSTON
Medical and Surgical Journal

Established in 1828

Published by The Massachusetts Medical Society under the jurisdiction of the following-named committee:

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Subscription Terms: *$6.00 per year in advance, postage paid for the United States, $7.56 per year for all foreign countries belonging to the Postal Union.*

Material for early publication should be received not later than noon on Saturday. Orders for reprints must be sent to the Journal office, 126 Massachusetts Ave.

The Journal does not hold itself responsible for statements made by any contributor.

Communications should be addressed to The Boston Medical and Surgical Journal, 126 Massachusetts Ave., Boston, Mass.

COMPULSORY INDUSTRIAL ACCIDENT INSURANCE

In the issue of Sept. 16, 1926, the legislative resolve providing for a committee to study the law relating to compulsory accident insurance was referred to.

The Workmen's Compensation Act, making provision for the payment of compensation to injured employees and certain provisions for the payment of medical and hospital fees, has been in effect since July 1, 1912. It was a measure intended primarily to benefit the injured workman. In general the act has been most beneficial. In regard to many details complaints have been made. The vast number of proposed amendments to the act which are introduced each year in the Legislature is evidence of this fact. Largely in order to deal intelligently with this bulk of bills the Legislature at its last session provided for the appointment of a commission of five who should review the whole subject and make such recommendations as in their judgment are wise to better the existing act. This Commission has just been appointed by the Governor and the Governor has most fittingly named as one of its members an Ex-President of the Massachusetts Medical Society Dr. Samuel B. Woodward. It is fortunate that the medical profession is to be so ably represented in this matter because in so many ways the act directly concerns the physicians of the Commonwealth. There are of course many phases of the act which concern solely the business aspects. The matters of chief concern to the physician and surgeon deal with the payment for professional services to the injured workman.

Since the passage of the act there have been many radical changs in the industrial and business world. The laborer today is better paid than ever before. The capitalist has not as much wealth to distribute in charity. The changes are no where more plainly to be seen than in hospitals. Incident to these changes the feeling is constantly growing that the members of a hospital staff should be more adequately remunerated than has been the custom for many years. It is only natural that the physician should expect to be paid for services which benefit both employer and employee and which are covered by insurance.

The act as it is has been of enormous benefit to the medical profession in doing just this thing. There are, however, certain phases of the act which seem to many to be unfair.

First is the matter of payment for services rendered to ward patients. The rapid increase rendered in the charges made to ward patients has naturally made the members of many a staff feel that these patients no longer are on a charity basis.

It is to be remembered however, that conditions differ greatly in various localities and various hospitals. To take an extreme example if all patients hurt in industrial accidents and treated at the Haymarket Relief Station of the Boston City Hospital were to pay the surgeon treating them the position of resident surgeon in this hospital would be sought most eagerly for financial reasons. Conditions differ totally in the small community hospital. It has, therefore, been the policy of the Industrial Accident Board to support as far as possible the policies adopted by the Trustees of the various hospitals in reference to the payment of the staff members for the care of ward patients. The problem is thus local and one which may well be settled between the staff and the Trustees in each locality.

Many considerations must be borne in mind however upon this big problem of direct remuneration to members of hospital staffs. The rate that the insurance companies are allowed to charge under the compensation law is fixed by the insurance commissioner on the basis of payments made and losses incurred. The larger the payments and losses the higher the rate of insurance. The amount that employers of labor are obliged to pay is also fixed by the insurance

commissioner. The rate of compensation payable to injured workmen and their dependents is fixed by the Legislature. The medical costs in 1925 amounted to nearly two and one-half millions of dollars. The sum paid to injured workingmen and women amounted to a sum sightly in excess of four millions of dollars. That paid to dependents of fatally injured employees was approximately one million dollars.

Any increase in medical costs which result from the payment of fees to members of hospital staffs can only be met by either increasing premiums (which are already acknowledged to be high) to the employers in each community, or counteracting the effect of such payments by allowing lower fees to men who are not on hospital staffs. The effect of such action upon the Legislature in the matter of other important amendments to the medical provisions, affecting physicians and surgeons generally, must also be considered.

The second objection to the act from the viewpoint of the physician is that compensation for services rendered is limited to the period of two weeks "except in unusual cases." This provision has done several things. It has inevitably tended to make physicians charge as much as possible for the care during the first two weeks in a case in which they know that they must give their services without pay for many more weeks to follow. And this tendency is hardly checked by the fact often overlooked that before the act was passed no renumeration at all was often to be expected.

Fortunately the Industrial Accident Board in its interpretations of "unusual cases" has been liberal. Recently a court decision has narrowed the board's interpretation of these words.

In the past attempts have been made to amend the law so that payment would be made for professional services as long as was necessary to bring about the maximum of improvement which could reasonably be expected. This would be the ideal. It is probable that provision could be made which would prevent abuse of such an amendment.

A third matter of interest to the medical profession is whether or not the word "disease" should be added to the act. In other words, is it necessary to add after the words "personal injury" the word "disease" so that the act may provide expressly for the payment of compensation and medical treatment in all cases of both "personal injury and disease" arising out of the employment. This seems unnecessary, as the Industrial Accident Board, again liberal in its interpretation, has from the very beginning held that all diseases of employment due to personal injuries arising out of the employment are covered. The Supreme Court, although somewhat cautiously, has affirmed such decisions, making it plain however that the ground of affirmation is that the diseases for which compensation is sought must be due to personal injuries arising out of the employment. Therefore, decisions involving lead poisoning, tuberculosis, acceleration of pre-existing disease, hernia, and the like, have been held to be under the compensation law. Therefore, it seems unnecessary to add the word "disease" to the act.

Doubtless other phases of the act concern the medical profession. The interest of physicians to consider the points at issue must be aroused promptly. Hearings will be held by the Commission. It may not be possible for the JOURNAL to announce the dates of these hearings in time to be of service to our readers. If anyone wishes to be heard prompt information can always be obtained through the Industrial Accident Board at the State House.

There is now presented to the medical profession of this commonwealth the opportunity to present suggestions which will make better an act which has done much good. The force of the suggestions will depend in great part upon whether they are presented in a broad minded or a selfish manner. The opportunity is now open. It is probable that no other similar opportunity will arise for many years to come.

REFLECTIONS ON THE MASSACHUSETTS BOARD OF REGISTRATION IN MEDICINE

IN the Federation Bulletin for September 1926, Frederick C. Waite, Ph.D., Professor of Histology and Embryology, Western Reserve University, contributes an article in which certain criticisms of the Massachusetts Board of Registration in Medicine appear. The first statement is that state licensing boards share in the responsibility of tolerating six medical schools which are not recognized presumably by the council on medical education and most of the licensing boards. The remedy according to Dr. Waite may be found in the refusal of the boards to accept applicants who are graduates of these schools. This is a correct statement in part but does not apply to the Massachusetts board because this board would follow this suggestion if it could. The fault lies with the legislature which obliges the board to accept all graduates of legally chartered medical schools which give a four years' course, over protests which have repeatedly been made in the form of amendments presented or endorsed by the board.

In another place in this article Dr. Waite states, speaking of Massachusetts, it must expect to continue to have this dubious reputation so long as the actions of its licensing board makes that state the largest harbor in the country for the graduates of derelict medical schools. He then calls on the medical profession and the lay public to employ remedial measures. The first of this section is not true for the board has not been given discretion. It has appealed to the

Attorney General for advice and has been told that it must, under the provisions of the law, accept the graduates of these schools. The later appeal to the medical profession and the lay public is sound. The board has done this repeatedly and forcefully.

A pointed criticism made by Dr. Waite is to the effect that some "licenses have been secured through corruption of public officials." If Dr. Waite knows of specific instances of corruption he is lacking in courage and laying himself open to the suggestion that he is adopting the custom of the blackguard in leaving it to be assumed that Massachusetts may have had corrupt members of the licensing board. He virtually repeats this charge later on. If he has evidence other than that cited with respect to Illinois he should give it. He tries to save his face by stating that the accusations are general rather than specific and only occasionally worthy of credence. If he believes that his accusations are true let him say what he means and cite specific instances and not by inference try to blacken the character of honorable public servants.

He has further reflections on the boards in the claim that members are being deceived by the applicants during an examination. He also criticises the questions submitted, the rating of the answers and the generosity of the examiners.

Many of his deductions based on the fact that an unreasonable number of graduates of low grade schools are able to secure recognition are sound but he fails to apply the proper explanation which is that in Massachusetts for example, the legislature expects the board to perform a difficult task without making adequate provision for the necessary machinery.

The legislature is confronted with the problem of dealing with a technical procedure of which the majority have no real understanding. In Massachusetts a considerable proportion of the legislators appear to believe that the recommendations with respect to better laws are based on a desire to strengthen a medical trust and restriet the practice to the product of the large universities. The argument in favor of the poor boy finds enthusiastic response in the hearts of some. Until the legislature can be led to endorse the recommendations of the board of registration, present conditions will continue to exist.

If Dr. Waite will come to Massachusetts and get acquainted with the members of the board he will find them honorable public servants anxious to serve the Commonwealth to the limit of the power conferred and not indifferent time servers. His criticism of the State, the laity, and to some extent the profession at large is unfortunately true to a considerable degree but he has no right to assume that the members of the board are not qualified for the work. They are not stupid, indifferent or mercenary. They are trying to do the best that can be done under the serious handicap of inhibitory laws. Great progress has been made under the operation of the Massachusetts Board and we can confidently assure the critics of Massachuetts that the average medical service is better in this state than is to be found in many states which have better laws. We have one standard of medical practice and do not license the cults as such.

REFLECTION ON DOCTORS

Captain George A. Parker, federal prohibition administrator for Massachusetts, in a speech before a Rotary Club a few weeks past is reported to have said that "the efforts of the prohibition force in this state are disrupted by the unscrupulous issuance of liquor prescriptions by many physicians." He claimed, according to the report, that 85,000 gallons of whiskey have been prescribed by physicians in Massachusetts last year. He modified the severity of his indictment by saying that the privilege of prescribing alcoholic beverages is not abused by honorable professional men.

It would be of interest to know how large a proportion of this 85,000 gallons was prescribed by honorable doctors for this would indicate the confidence of physicians in the therapeutic value of whiskey.

Since Mr. Parker believes that the doctors are largely to blame for the present day conditions, it is remarkable that so few doctors have been called before the Board of Registration in Medicine on charges of unethical prescribing of liquors. We are quite sure that irregularities pertaining to the practice of medicine will be corrected if good evidence is presented to the Board. If there is reason to believe that doctors are so unethical and antagonistic to the law it would seem that more should have been called before the Board.

The customs and habits of people cannot be changed in the space of a few years and honorable physicians may be justified in many instances in issuing prescriptions to those persons who are mildly ill and who have been in the habit of using alcoholic beverages. To withhold alcohol in some cases may cause trouble.

We are quite sure that physicians are as careful in observing the prohibition requirements as any other group of persons and we confidently believe that failure to enforce the law depends more on some of the unreasonable and impracticable features of the act coupled with indisposition of the government to strictly enforce the provisions of law rather than to the actions of physicians.

A considerable number of doctors haven't taken out permits, many who have permits are honorable and we doubt the wisdom of putting the blame on the medical profession. The subjoined letter is pertinent.

Board of Registration in Medicine
Room 144, State House, Boston

August 19, 1926.

Editor, Boston Medical and Surgical Journal:

In reply to your letter of August 18, relative to statements made by Captain Parker, Federal Prohibition Administrator, you are informed that for 1925 and 1926 to date, we have received twenty-one reports from Captain Parker—all liquor permits for physicians, which have been revoked by his department.

Of this number, eighteen have been acted upon since January, 1925, and three are now pending—one of which we are holding on advice of the Prohibition Department.

Yours very truly,
FRANK M. VAUGHAN, *Secretary.*

THIS WEEK'S ISSUE

CONTAINS articles by the following authors:

LAWRENCE, JOHN S. A.B.; M.D. Medical School of University of Virginia 1921. Edward Hickling Bradford Fellow in Medical Research, Harvard Medical School. His address is Medical Department, Vanderbilt University, Nashville, Tennessee. Associated with him is

BOCK, A. V. A.B.; M.D. Harvard Medical School 1915. Associate physician Massachusetts General Hospital; Assistant Professor of Medicine, Harvard Medical School; Member American Society for Clinical Investigation; Associate member The Association of American Physicians. Address: Massachusetts General Hospital. They write on "Relationship Between Gastric Ulcer and Carcinoma of Stomach." Page 651.

CARTY, JOHN RUSSELL. B.S.; M.D. Cornell University Medical College 1921. Chief of Clinic of Roentgenology and Instructor of Roentgenology Cornell Medical College; Member New England Roentgen Ray Society. The title of his paper is "Diagnostic Significance of Lateral Curvature of the Spine Caused by Muscular Spasm". Page 654. Address: 28th Street and First Avenue, New York City.

DRURY, DANA W. M.D. Medical School of Harvard University 1904. F.A.C.S. Associate Aural Surgeon Massachusetts Eye and Ear Infirmary; Aurist, Boston Floating Hospital. His subject is "Dehiscence of the Floor of the Middle Ear". Page 657. Address: 416 Marlborough Street, Boston.

PARSONS, ELOISE. Ph.D.; M.D. Rush Medical College 1924. Associate Professor in Physiological Chemistry University of Chicago, Fellow in Medicine Mayo Foundation. The subject of this paper is "Benign Glycosuria with Hyperglycemia: Report of Case with Metabolic Studies." Page 660. Address: Mayo Clinic, Rochester, Minn.

SMITH, MILLARD. B.S.; M.S.; M.D. Harvard Medical School 1923. Assistant in Thorndike Memorial Laboratory, Boston City Hospital; Assistant in Medicine Harvard Medical School; Member American Chemical Society. The title of the paper is "An Unusual Blood Sugar Finding During an Insulin Reaction." Page 663. Address: 329 Longwood Avenue, Boston.

HUBER, EDWARD G. Detailed record on page 291, No. 6, Vol. 195. His article is a continued account of "The Control of Communicable Diseases in Massachusetts." Page 665. Address: The War Department, Washington, D. C.

HOWARD, HERBERT H. B.S.; M.D. Harvard Medical School 1912; Assistant Visiting Physician, Boston City Hospital; Member American Urological Association. He writes on "Progress in Urology." Page 673. Address: 395 Commonwealth Avenue, Boston.

RECENT DEATHS

BROWNRIGG—DR. JOHN SYLVESTER BROWNRIGG, identified with the Boston Department of Health for many years, died September 21, 1926, at his home in Roxbury at the age of 69.

Born in Boston August 22, 1857, the son of John and Jane (McGruder) Brownrigg, he was graduated from Boston College and took his degree from Harvard Medical School in 1887. After some time in practice, Dr. Brownrigg was made a member of the City Health Department, and in 1921 was promoted to be chief medical inspector. He retired in August, 1924.

Dr. Brownrigg was always interested in the work of the Carmelite Convent on Mt. Pleasant Avenue, Roxbury, donating his professional services there; and also in the work carried on by the Mission Church in Roxbury. He was a member of the American Medical Association and of the Massachusetts Medical Society and an associate member of the Boston Medical Library.

Dr. Brownrigg, who was a widower, leaves a son, William Brownrigg, and a daughter, Miss Jane T. Brownrigg, both of this city.

PILLSBURY—DR. GEORGE HARLAN PILLSBURY, a retired member of the Massachusetts Medical Society, died at his home in Lowell, as a result of injuries received in an automobile accident, September 16, 1926, aged 83.

Dr. Pillsbury's date of birth was June 8, 1843. The public schools of Lowell gave him his elementary education, after which he entered Dartmouth College and was graduated in 1865. In the following year he entered Harvard Medical School, where he received his dgeree of M.D. in 1869. Later he took a postgraduate course at the Ecole de Medicine in Paris. When ready to begin active work in his profession he opened offices in Lowell and soon built up an extensive practice. He manifested interest in all civic affairs and served for a time as a member of the School Committee. While it may not be said that Dr. Pillsbury was a motoring enthusiast, he traveled about almost exclusively in a car that he operated himself and gave no apparent indication that his advanced years were at all a drawback in its successful operation.

On June 5, 1872, Dr. Pillsbury married Mary A. Boyden, who survives him, with four children, Dr. Boyden H. Pillsbury of Lowell, Major Harry C. Pillsbury and Colonel George B. Pillsbury, both of the United States army, and Mary B. Pillsbury, librarian

at Vassar College. He also leaves a brother, Samuel L. Pillsbury of Columbus, Ohio. He was a member of St. Anne's Episcopal Church and of Kilwinning Lodge, A. F. and A. M.

CONNECTICUT DEPARTMENT OF HEALTH

MORBIDITY REPORT FOR THE WEEK ENDING SEPTEMBER 4, 1926

Diphtheria	9	Chickenpox	2
Last week	10	German measles	1
Diphtheria bacilli carriers	3	Influenza	1
Typhoid fever	8	Malaria	1
Last week	4	Mumps	1
Scarlet fever	13	Paratyphoid fever	1
Last week	8	Pneumonia, lobar	6
Measles	9	Poliomyelitis	1
Last week	10	Septic sore throat	18
Whooping cough	36	Tuberculosis, pulmonary	33
Last week	21	Tuberculosis, other forms	2
Bronchopneumonia	7	Gonorrhea	66
Cerebrospinal meningitis	1	Syphilis	32

MORBIDITY REPORT FOR THE WEEK ENDING SEPTEMBER 11, 1926

Diphtheria	8	Chickenpox	2
Last week	9	Dysentery, bacillary	1
Diphtheria bacilli carriers	3	Encephalitis, epidemic	2
Scarlet fever	19	German measles	1
Last week	13	Mumps	1
Typhoid fever	9	Paratyphoid fever	1*
Last week	8	Pneumonia, lobar	10
Measles	6	Poliomyelitis	3
Last week	9	Septic sore throat	1
Whooping cough	23	Tuberculosis, pulmonary	11
Last week	36	Gonorrhea	10
Bronchopneumonia	4	Syphilis	8
Cerebrospinal meningitis	2		

*Delayed reports.

REPORTS AND NOTICES OF MEETINGS

NINTH ANNUAL MEETING OF THE AMERICAN DIETETIC ASSOCIATION

THE ninth annual meeting of the American Dietetic Association will be held October 11, 12, 13, 1926, at the Ambassador, Atlantic City, N. J. Detailed program may be obtained by writing to Mrs. Dorothy K. Hassler, Riverside, Ill.

MASSACHUSETTS STATE NURSES ASSOCIATION

THE Autumn meeting of this Association will take place at the Hotel Bancroft, Worcester, October 8 and 9.

The address of welcome will be delivered by Hon. M. J. O'Hara, mayor of Worcester.

The several educational and ethical problems relating to nursing will be covered by speeches to be presented by leading members of the Association.

Addresses will also be delivered by Drs. Kendall Emerson, Thomas F. Kenney, George H. Bigelow and Ernest L. Hunt. A very attractive program has been arranged.

NEW ENGLAND SURGICAL SOCIETY

PROGRAM OF THE ANNUAL MEETING AT BOSTON, MASS., OCTOBER 1 AND 2, 1926

Headquarters—Hotel Copley Plaza.

Presentation of papers will be limited to 15 minutes. Discussion to five minutes for each speaker. All manuscript should be presented to the Recorder promptly.

All papers will be published in the BOSTON MEDICAL AND SURGICAL JOURNAL, the official organ of the Society.

GENERAL PROGRAM

Thursday, September 30

7 P. M.—Meeting of the Executive Committee, Harvard Club.

Friday, October 1

9 A. M.—Clinic, New England Deaconess Hospital.
10.30 A. M.—Clinic, Peter Bent Brigham Hospital.
1 P. M.—Luncheon, Harvard Medical School.
2 to 4 P. M.—Literary Program, Harvard Medical School.

1. Obscure Lesions of the Shoulder. Dr. E. A. Codman, Boston, Mass.
2. Life Saving Details. Dr. Frederick B. Sweet, Springfield, Mass. Discussion opened by Dr. A. M. Rowley, Hartford, Conn.; Dr. P. E. Truesdale, Fall River, Mass.
3. The Operative Treatment of Fractures. Dr. Charles L. Scudder, Boston, Mass.
4. A Short Résumé of Fracture Experiences at the Norton Company. Dr. W. I. Clark, Worcester, Mass.
5. Rupture of Kidney. Dr. P. E. Truesdale, Fall River, Mass.
6. Staphylococcus Spinal Meningitis with Operation and Recovery. Dr. Kendall Emerson, Worcester, Mass.
7. Operative Experiences with Eight Cases of Oesophageal Diverticulum. Dr. F. H. Lahey, Boston, Mass.

4.30 P. M.—Reception to members of the Society at the home of Dr. Charles G. Mixter, 57 Clyde Street, Brookline, Mass.

7.30 P. M.—Annual dinner at the Harvard Club.

1. President's address.
2. Diabetic Surgery from a Medical Point of View. Dr. Elliott P. Joslin, Boston, Mass.

Saturday, October 2

9 A. M.—Clinic, Boston City Hospital.
11 A. M.—Clinic, Massachusetts General Hospital.
1 P. M.—Luncheon, Massachusetts General Hospital.
2 P. M.—Executive session, Old Amphitheatre, Massachusetts General Hospital.
2.30 P. M.—Literary program. Remarks on the History of the Old Surgical Amphitheatre. Dr. F. A. Washburn, by invitation.
8. Pyloric Stenosis. Dr. W. E. Ladd, Boston, Mass.
9. Carcinoma of the Stomach. Dr. Edward R. Lampson, Hartford, Conn.
10. Acute Massive Hemorrhage from the Stomach and Duodenum. Dr. David Cheever, Boston, Mass.
11. The Mortality Rate of Appendicitis—Is It on the Increase? Dr. Arthur W. Marsh, Worcester, Mass. Discussion opened by Dr. Lyman Allen, Burlington, Vt.; Dr. Thomas D. Luce, Portsmouth, N. H.
12. Fibromatosis of Sigmoid. Dr. Peer P. Johnson, Beverly, Mass.
13. Caesarean Section After Death of the Mother. Report of Two Cases. Dr. M. F. Fallon, Worcester, Mass. Discussion opened by Dr. W. P. Graves, Boston, Mass.
14. The Small Obstructing Prostate. Dr. A. L. Chute, Boston, Mass.